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CONTRIBUTORS

FEBRUARY ISSUE

HARRY BAKWIN, M.D., F.A.A.P. Professor of Clinical Pediatrics, New York University-Bellevue Medical Center; Visiting Physician, Children's Medical Service, Bellevue Hospital; Attending Pediatrician, University Hospital, New York City.

GLADYS S. BENZ, M.A., R.N. Chairman, Department of Maternal and Child Nursing, State University of Iowa, Iowa City; Formerly Instructor in Pediatric Nursing, Children's Hospital, Cincinnati.

CHARLES BRADLEY, M.D., F.A.A.P. Associate Professor of Pediatrics and Psychiatry, University of Oregon Medical School, Portland.

LAWRENCE CHAFFIN, M.D., F.A.C.S. Clinical Professor of Surgery, University of Southern California Medical School; Senior Surgeon, Los Angeles County Hospital and Los Angeles Children's Hospital; Staff, Hospital of the Good Samaritan, Los Angeles.

ANTHONY L. DANZA, M.D. Assistant Instructor in Surgery, Albert Einstein College of Medicine; Chief Resident, Thoracic Surgery, Albert Einstein Hospital, New York City.

DONALD A. DAVIS, M.D., F.A.C.S. Associate Professor of Clinical Surgery, New York University-Post Graduate Medical School; Associate Visiting Surgeon, Bellevue and Gouverneur Hospitals; Associate Attending Surgeon, University Hospital, New York City.

ERNEST CARROLL FAUST, M.A., PH.D. William Vincent Professor of Tropical Diseases and Hygiene, Tulane University School of Medicine, New Orleans; Consultant, Armed Forces Institute of Pathology, Washington, D.C.; Member of Expert Committee on Parasitic Diseases, World Health Organization; Member of Committee on Revision, U.S. Pharmacopeia.

JOHN H. JUHL, M.D. Associate Professor of Radiology, University of Wisconsin Medical School; Radiologist, University Hospitals, Madison.

RODNEY C. JUNG, M.D., PH.D. Assistant Professor of Tropical Medicine, Tulane University School of Medicine; Visiting Physician, Charity Hospital of Louisiana; Clinical Assistant in Gastroenterology, Touro Infirmary, New Orleans; Visiting Physician, Illinois Central Hospital, Chicago.

EDWARD A. KAMENS, M.D. Resident Surgeon, Bronx Municipal Hospital Center (Albert Einstein College of Medicine), New York City.

SAMUEL KARELITZ, M.D., PH.D., F.A.A.P. Associate Professor of Clinical Pediatrics, Columbia University College of Physicians and Surgeons; Chief of Pediatrics, Long Island Jewish Hospital; Attending Pediatrician, Mt. Sinai Hospital, New York City.

C. EVERETT KOOP, M.D., Sc.D. (Med.), F.A.C.S., F.A.A.P. Associate Professor of Pediatric Surgery, University of Pennsylvania School of Medicine and Graduate School of Medicine; Surgeon-in-Chief, Children's Hospital of Philadelphia.

BARBARA LIPTON, M.D. Anesthesiologist (Adjunct), Beth Israel Hospital, New York City.

ALBERT S. LYONS, M.D., F.A.C.S. Adjunct Attending Surgeon and Chief, Intestinal Rehabilitation Clinic, Mt. Sinai Hospital, New York City.

CLAUDE G. MENTZER, M.D., F.I.C.S., F.A.PR.S. Clinical Assistant Professor in Proctology, University of Miami School of Medicine; Attending on Proctologic Service, Jackson Memorial Hospital; Attending-Chief of Proctology, Victoria Hospital, Miami.

DAVID W. MOSS, M.D. Resident in Surgery, Mount Sinai Hospital, New York City.

LESTER W. PAUL, M.D., F.A.C.R. Professor of Radiology, University of Wisconsin Medical School; Radiologist, University Hospitals, Madison.

WILLIS J. POTTS, M.D., F.A.C.S. Professor of Pediatric Surgery, Northwestern University Medical School; Surgeon-in-Chief, Children's Memorial Hospital, Chicago.

BENJAMIN G. P. SHAFIROFF, M.D., F.A.C.S. Associate Professor of Clinical Surgery, New York University College of Medicine; Chief Surgeon, Caledonian Hospital; Director of Surgery, Goldwater Memorial Hospital and Brooklyn Women's Hospital, Brooklyn.

MONICA H. SNYDER, M.D. Courtesy staff, Los Angeles County Hospital, Los Angeles, and St. Joseph's Hospital, Burbank, California.

WILLIAM H. SNYDER, JR., M.D., F.A.C.S. Associate Professor of Surgery, University of Southern California School of Medicine; Chief of Surgical Service, Los Angeles Children's Hospital; Chief of Pediatric Surgical Service, Los Angeles County General Hospital; Senior Surgeon, Hollywood Presbyterian Hospital; Active Staff, St. Joseph's Hospital, Los Angeles.

ROBERT TURELL, M.D., F.A.C.S. Assistant Professor of Surgery, in Charge of Proctology, Albert Einstein College of Medicine; Associate Surgeon, in Charge of the Colon and Rectal Service, Harlem Hospital; Associate Surgeon (Proctology), Montefiore and Beth Israel Hospitals; Associate Surgeon (Proctology), and Chief of Rectal Clinic, Mt. Sinai Hospital; Attending Surgeon, Bronx Municipal Hospital Center. New York City.

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CORRECTION

The second sentence of the fourth paragraph on page 135 of the February, 1955, number of the PEDIATRIC CLINICS OF NORTH AMERICA should read as follows: "In lower Burma the levels of sensitivity are 14 to 27 per cent, while in upper Burma the levels are 4 to 8 per cent, depending on the age group."

FOREWORD

ROBERT TURELL, M.D.

To be uncertain is to be uncomfortable.—*Chinese proverb.*

At the present time pediatric proctology is still considered by some, to cite Sir Winston Churchill, to be a riddle wrapped in a mystery inside an enigma. The purpose of this symposium is to emphasize that the juvenile patient is not only heir to most of the morbid proctologic processes encountered in the adult patient, but is also subject to the effects of congenital malformations. It is of course now well realized that the care of sick juveniles demands special care of the patient as well as of the disease, for they are not just diminutive adults.

This symposium is primarily addressed to the pediatrician (and the general practitioner), who is the first to see the patient and upon whose health advice the parents of the patient rely. He is the important intermediary between the proctologist and the parents and is frequently called upon to pass judgment on the management recommended by the proctologic surgeon. An attempt has been made to concentrate and correlate recent theories and developments in one place. The subject of proctologic disease has been approached from a broad viewpoint. Controversial and, on occasion, even questionable ideas are included as long as they are stimulating or provocative. To accomplish this task I invited a distinguished group of authors and teachers to write concise and clear articles of practical value.

My task in arranging this symposium consisted primarily in the preservation of objectivity and in the prevention of undue overlapping. My genuine gratitude goes to all contributors of this symposium. My thanks go to the publishers and their staff for their splendid cooperation and for the opportunity to serve as Consulting Editor.

APPLIED ANATOMY OF THE COLON AND RECTUM

B. G. P. SHAFIROFF, M.D.

The structures that comprise the large intestine in its collective sense (cecum, ascending colon, transverse colon, descending colon, sigmoid and rectum) and their associate supporting structures are involved in a variety of processes which stimulate the interest of all concerned with the treatment of infants and children. In this paper several subjects of interest are presented on an anatomic basis for practical application in clinical pediatrics. These subjects are (1) the variational anatomy of the colon, (2) mesenteric cysts and retroperitoneal hernia in relation to the colon, (3) the lymphatic drainage of the colon, (4) the venous return of blood from the rectum, (5) the autonomic nervous system to the colon and rectum, (6) the sphincters of the rectum, (7) congenital anomalies of the rectum.

THE VARIATIONAL ANATOMY OF THE COLON

Wide variations are possible in the form, position and relation of the colon to other abdominal viscera. Alterations from the normal may lead to gastrointestinal complaints and symptoms early in infancy and childhood. The cecum is the pouchlike portion at the beginning of the large intestine marking the junction between colon and ileum. It usually occupies the right iliac fossa, overlying in this area the iliopsoas muscle and fascia. Its position, however, may vary, depending on the degree of arrest of the mesentery of the cecum during any part of its descent or on the possibility of the persistence or absence of the mesentery. Variations of the latter type cause abnormal placement or

From the Department of Surgery, New York University College of Medicine, New York City.

result in preternatural mobility of the cecum and ascending colon. Under normal conditions the cecum and colon become fixed to the posterior parietal peritoneum with resorption of their mesenteries.

Variations in the process of mesenteric fusion and fixation result also in the formation of abnormal intra-abdominal bands or membranes. Abnormal developmental bands in the region of the cecum and appendix such as Lane's ileal membrane may cause increased tension on the mesentery, spasm of the ileocecal sphincter, ileal stasis, strain on the ileocolic vessels and stimulation of the autonomic nerves locally.

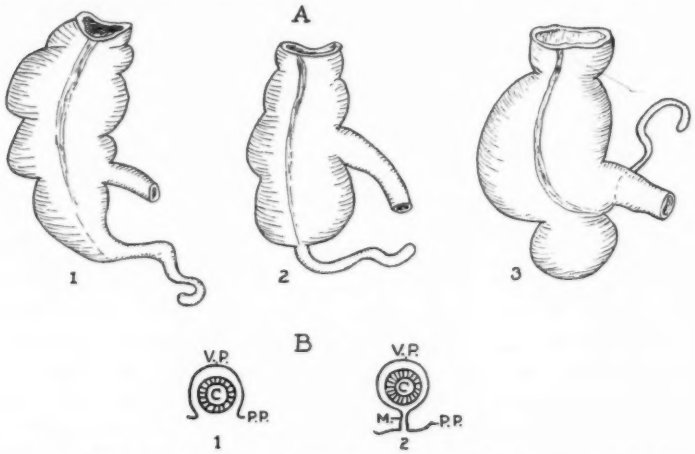

Fig. 1. *A*, Various shapes assumed by the cecum after development: *1*, fetal type; 2, incomplete development of cecum; 3, normal cecum. *B*, Cross section of normal cecum (*1*) and cecum with congenital persistence of mesentery (*2*): *P.P.*, posterior parietal peritoneum; *M.*, congenital mesentery of cecum; *V.P.*, visceral peritoneum.

Lane's membrane is a triangular or quadrilateral membrane attached externally to the parietal peritoneum of the iliac fossa and internally to the antimesenteric border of the ileum. It is due to the low descent of the fusing membranes of the mesentery of the colon with the dorsal parietal peritoneum and includes the mesentery of the terminal ileum. *Periappendicular membranes* usually originate from the mesentery of the cecum, appear early in the third month of development, and may prevent the proper fusion of the mesentery of the ascending colon. *Jackson's membrane* is a thin vascular membrane which extends over the middle third of the ascending colon and reaches anteriorly to the longitudinal muscular band. *Mesocolic jejunal membranes* are irregular developmental fusions which occur after rotation of the colon when the mesocolon takes its position across the duodenum and extend from the under surface of the mesocolon to the antimesenteric surface of the

first part of the jejunum. *Right and left intercolic membranes* represent downward extensions of the omental bursa which overlap and involve the upper portions of the ascending and descending colon and thus cause sharp angulation at these points.

Abnormal early commencement of fusion areas in fetal life as well as abnormal extension downward of normal fusion areas is causative of untoward mobility or prolapse of the ascending colon and cecum which results in a symptom complex of ptosis and stasis.

Susceptibility of the cecum to distention favors inspissation of intestinal contents, gaseous pocketing and retention of fecal and foreign body concretions. The interior of the cecum is furrowed by the orifices

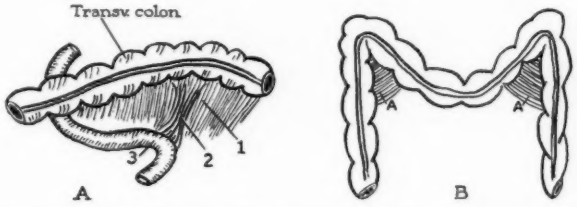

Fig. 2. Developmental membranes of transverse colon. *A*, Mesocolic jejunal membrane: *1*, transverse mesocolon; *2*, mesocolic jejunal membrane; *3*, duodenojejunal flexure. *B*, Intercolic membrane (A-A).

of the appendix and ileum. The opening of the ileum into the junction of colon and cecum is marked by two strong folds, an upper and a lower fold which traverse obliquely the greater part of the interior of the ileocecal valve. The valve itself is fixed into the cecum by a strong retinaculum on its mesial and lateral aspects. In the fetal type of cecum the internal opening of the appendix is usually unguarded, while in other types variations from a simple mucosal fold to a true valvelike structure can be seen. It should be noted that at birth the cecum is located higher than in the adult because it has not descended to its permanent position and adhesion of the mesentery of the ascending colon to the parietal peritoneum has not been completed. At birth and in the first three to five years of life the cecum is often of the fetal type and is small in size, while the appendix is relatively large.

MESENTERIC CYSTS AND RETROPERITONEAL HERNIA IN RELATION TO THE COLON

Mesenteric cysts occur only between the layers of the mesentery of the colon and small intestine, while the term "retroperitoneal cysts" should be reserved for those cysts situated in the retroperitoneal tissues without connection to any structure except by areolar tissue. True mesocolic cysts are of mesocolic origin and are usually found near the

terminal part of the ileum and occasionally in other areas below the transverse mesocolon between the ascending and descending mesocolon. These cysts have three possible origins: (1) from mesodermal remnants incarcerated behind the developing peritoneum with final migration of the remnants between the mesenteric layers, (2) cysts of intestinal origin as sequestration diverticula from the bowel, and (3) persistent portions of vitelline duct finally undergoing cystic degeneration. Two other types of cysts may be encountered within the proximity of the colonic tract: (1) cysts arising in cell inclusions such as the dermoid of teratomatous origin and (2) the lymphatic cyst originating from the lymphatics of the intestine.

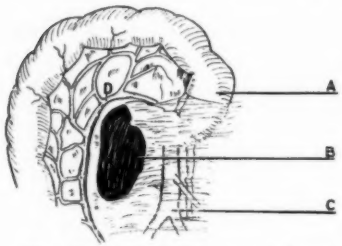

Fig. 3. Diagram of mesenterico-parietal fossa. A hernia in this fossa occupies the right half of the abdomen. *A*, Beginning of jejunum; *B*, mesenterico-parietal fossa; *C*, aorta; *D*, superior mesenteric artery. (After Short.)

Retroperitoneal hernia occurs in spaces resulting from abnormal development of the parietal peritoneum. Herniation of bowel through these unprotected areas may cause recurrent subacute intestinal obstruction or acute strangulation. In children it is possible to palpate the hernial mass as a tumor.

Besides the usual paraduodenal fossae and the uncommon right duodenojejunal fossa, also called the mesenterico-parietal fossa, there are four other potential sites for retroperitoneal herniation affecting the colonic tract: (1) the *retrocolic fossa*, which lies between the cecum and ascending colon with its orifice directed downwards; (2) the

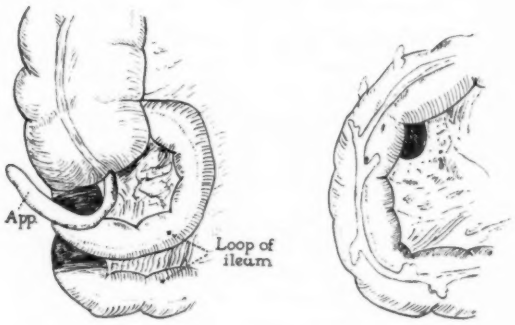

Fig. 4. A, Diagram showing obstruction caused by internal herniation into the retrocolic fossa. (After Pribram.) *B*, Diagram of intersigmoid fossa formed by the attachment of the pelvic mesocolon. (After Short.)

ileoappendicular fossa, which lies between the ileoappendicular fold and the mesentery of the appendix with its orifice pointing to the left and down; (3) the *ileocecal fossa,* bounded anteriorly by the ileocecal fold, posteriorly by the mesentery of the appendix and superiorly by the posterior surface of the ileum; and (4) the *intersigmoid fossa,* associated with mesosigmoid development and resulting in a developmental defect over the bifurcation of the left common iliac artery.

THE LYMPHATIC DRAINAGE OF CECUM AND COLON

The lymphatic drainage of the proximal colon involving the terminal ileum, appendix and cecum is of clinical interest because the lymph pathways from these organs can account for upper abdominal complaints ordinarily referable to the gallbladder, stomach or duodenum. The lymphatic drainage of the ileocecal region is richer than that of the rest of the colon. The ileocolic chain of lymph nodes often reaches 20 in number and consists of the anterior ileocolic, posterior ileocolic,

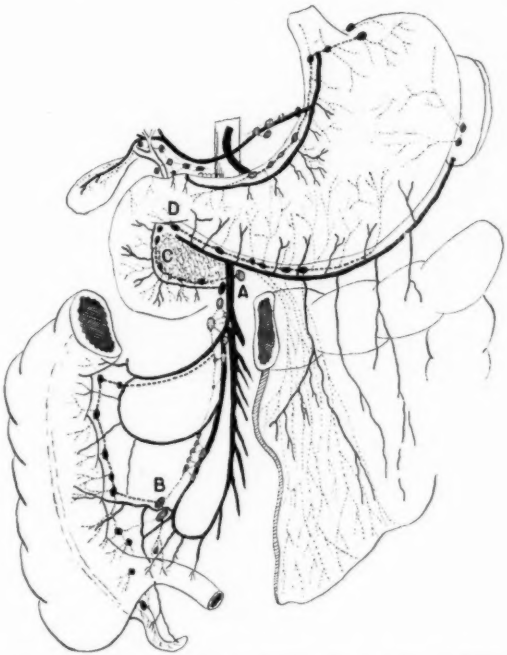

Fig. 5. Possible avenues of extension of lymph node drainage from the lymph nodes located in the cecum. *A,* Lymph nodes around the superior mesenteric artery; *B,* cecal lymph nodes; *C,* pancreaticoduodenal lymph nodes; *D,* subpyloric lymph nodes. (After Brathwaite.)

appendicular, iliac and right colic group of lymph nodes. From the paracolic lymph nodes located along the bowel wall, five or six efferent lymphatic ductules pass through one or more chains of lymph nodes and follow the course of the ileocolic artery to its origin from the superior mesenteric artery. As a result, the regional termination of lymph drainage occurs in front of the third segment of the duodenum into the lymph nodes around the superior mesenteric artery and from there into the receptaculum chyli. Lymphatic drainage from the cecum and appendix may also be diverted to the lymph nodes along the concavity of the duodenum (pancreaticoduodenal lymph nodes) and then to the subpyloric nodes, hepatic nodes and finally to the receptaculum via the celiac nodes. Another accessory pathway is provided by the omentum attached to a diseased viscus in the right lower quadrant of the abdomen. In this case the lymph flow is directed to the inferior gastric lymph nodes situated along the right half of the greater curvature of the stomach, from which point it is directed to the subpyloric lymph nodes.

Lymphatic drainage by the anatomic pathways described may cause irritative phenomena in distant organs. Moynihan, Brathwaite and others have described a syndrome of reflex appendicular dyspepsia and chronic duodenal ileus and stasis due to lymphatic extension from primary disease in the appendix or cecum. It is interesting that lymphatic communications between the appendix and such pelvic viscera as tubes, ovaries, prostate and so on have not been demonstrated.

THE VENOUS RETURN OF BLOOD FROM THE RECTUM

The venous drainage of the distal colon is also of clinical interest because of the major venous tributaries carrying blood from this area. The *superior hemorrhoidal venous plexus* (internal) commences in the submucosa of the rectum above the anorectal line and increases in size until the point of perforation through the midrectum. The plexus continues on the outer coat of the rectum and there forms the superior hemorrhoidal vein joining the inferior mesenteric vein with the other colic tributaries to drain finally into the splenoportal system. Inflammatory diseases of the rectum involving this venous system may cause thrombophlebitis and embolic phenomena directed towards the liver, especially because these veins are usually unprotected by valves.

The *inferior hemorrhoidal plexus* (external) originates below the anorectal line, principally around the anal canal below the levator ani. From the latter originate (1) the *middle hemorrhoidal vein*, which drains into the internal iliac vein; (2) the *inferior hemorrhoidal vein*, which drains into the internal pudendal branch of the internal iliac vein; and (3) the *middle sacral vein*, which drains into the left com-

mon iliac vein. The latter tributaries have an effective valvular system. Inflammatory involvement in this plexus is therefore transferable to

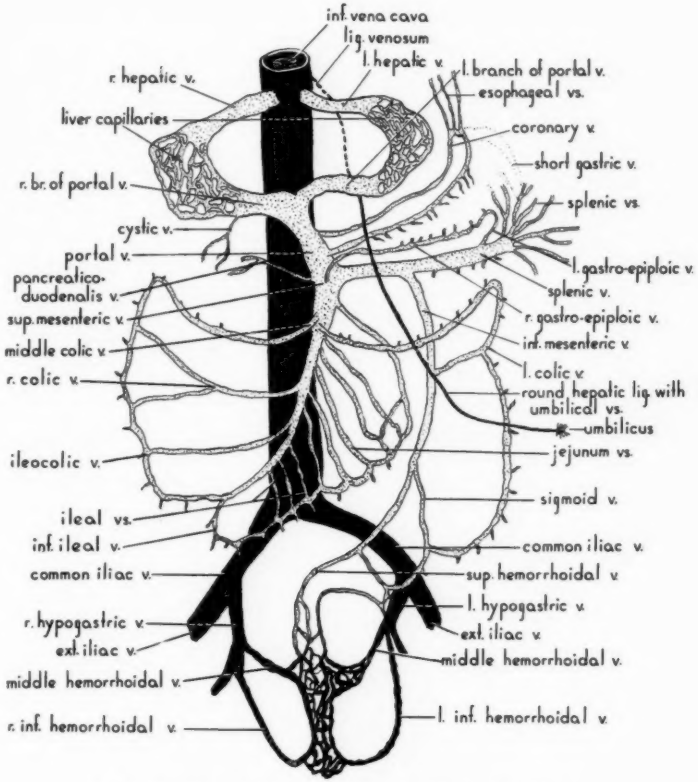

Fig. 6. Diagram of abdominal visceral portacaval venous system.

the iliac veins and the inferior vena cava. Inflammatory pathology involving the rectum and anus and distant reactions resulting therefrom can be predicted on the basis of these anatomic facts.

AUTONOMIC NERVOUS SYSTEM OF COLON AND RECTUM

Knowledge of the anatomy of the autonomic nerve supply to the colon and rectum is of importance in the study of diseases such as megacolon, ulcerative colitis and other dysfunctions of defecation. The neural apparatus to these structures comprises a complicated system of extrinsic and intrinsic nerves functionally related. The visceral auto-

nomic nerves (extrinsic) reach the cecum, the ascending colon and the proximal half of the transverse colon via the superior mesenteric plexus, which provides a periarterial pathway of intestinal distribution for both the vagus (parasympathetic) and sympathetic nerves. The superior mesenteric ganglion itself is a continuation of the celiac plexus and contains the postganglionic fibers of the dorsal sympathetic nerves and fibers from the upper lumbar chain.

The distal colon is innervated by the sympathetic fibers derived mainly from the lumbar section of the sympathetic chain (lumbar colonic nerves). These branches of communication from the right and left sympathetic chains unite in front of the aorta together with fibers from above to form the preaortic plexus, which descends over the abdominal aorta as far down as the inferior mesenteric artery and the iliac bifurcation. The inferior mesenteric artery and its distributing branches are encircled by the nerves of the inferior mesenteric plexus which come off from the preaortic plexus. Periarterial fibers ascend with the upper branch of the left colic artery to reach the splenic flexure; other fibers descend with the sigmoidal arteries and the superior hemorrhoidal artery to enter the sigmoid colon and the rectum.

The preaortic plexus near the level of the promontory of the sacrum consolidates to form the compact superior hypogastric plexus, which promptly splits into the right and left inferior hypogastric plexuses, also known as the right and left presacral nerves. The latter continue down over each of the common iliac arteries to lose their identity in a flat, irregular mass of nerve tissue which seems fixed bilaterally on the posterior and lateral aspects of the pelvis. These nerve structures are best designated as the right and left pelvic plexuses. Visceral fibers from the second and third sacral nerves and occasionally contributions from the fourth and fifth sacral nerves are traceable to the posterior aspect of the pelvic plexus. These are the parasympathetic components to the pelvic plexus and are usually designated as the nervi erigentes. The pelvic plexus like the celiac plexus in the upper abdomen contains both sympathetic and parasympathetic components through which the autonomic nerves are channelled in three peripheral divisions for distribution to the rectum, bladder and genital organs.

Short, rather thick fibers (four to six in number) from the rectal division of the pelvic plexus enter the rectum directly at the level of the plexus; other, thinner fibers (two or three in number) ascend through the mesosigmoid high up to the splenic flexure. These ascending nerve fibers hold no relationship to the colic vessels as do the sympathetic fibers of the inferior mesenteric plexus, which are periarterial. The colonic fibers of the pelvic plexus in their ascent through the mesosigmoid cross superficial to the vascular arcades and branch off to invest the colon as intestinal branches each a distance of about

2 inches from the others. The cell bodies of these parasympathetic fibers are located in the cord for the sacral outflow and are preganglionic. Through the urinary and genital divisions of the pelvic plexus, autonomic nerves are distributed to the bladder, uterus and so forth.

The intrinsic nervous system of the colon and rectum is contained within the intestinal wall as three well defined anatomic plexuses: (1) the subserous plexus, (2) the submucous plexus, and (3) the myenteric plexus. Auerbach's or the myenteric plexus is located between the inner circular and the outer longitudinal muscular coats. The ganglion cells of the plexus have been differentiated into three types of Dogiel cells plus a supporting group of interstitial cells of Cajal. This enteric plexus extends from the esophagus to the termination of the rectum beyond the internal sphincter.

Until recently the variation in the number of ganglion cells at various intestinal levels was of anatomic interest only. Now it has been definitely established that abnormal embryogenesis may result in agenesis of the enteric plexus in any part of the gastrointestinal tract. Absence of the enteric plexus in the colon is definitely established as causative of congenital megacolon. The vagus nerve terminates in the intestinal wall as preganglionic fibers completing the visceral reflex through the ganglion cells of the enteric plexus. The sympathetic branches terminate intramurally as postganglionic fibers in communication with smooth muscle cells, having previously completed a synapse in either a vertebral or prevertebral ganglion. The enteric plexus functions as a relay of stimuli to the intestinal musculature and in the coordination of these movements. Disruption of the continuity of peristaltic contractions occurs at the site

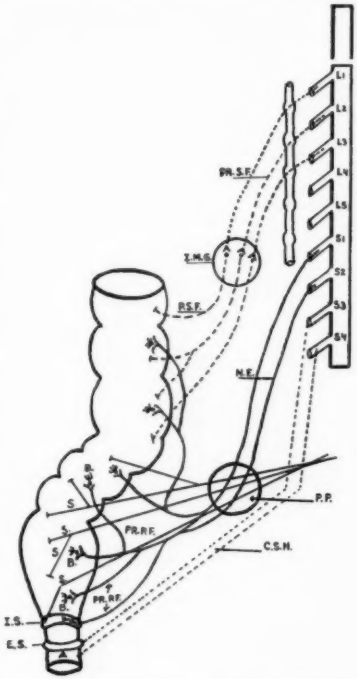

Fig. 7. Autonomic nervous system to descending colon. *I.M.G.*, Inferior mesenteric ganglion; *PR.S.F.*, preganglionic sympathetic fiber; *P.S.F.*, postganglionic sympathetic fiber; *N.E.*, nervus erigens; *P.P.*, pelvic plexus; *S.*, sympathetic fiber from hypogastric plexus; *PR.P.F.*, preganglionic parasympathetic fiber; *B.*, postganglionic parasympathetic fiber; *I.S.*, internal sphincter; *E.S.*, external sphincter; *C.S.N.*, cerebrospinal pudendal nerve to anus (A).

of agenesis of the enteric plexus. The involved segment is noncontractile except for limited myogenic rhythmicity, and although the distal sphincter is anatomically intact, this finely synchronated mechanism integrated with intestinal motility becomes disorganized and a primary functional obstruction gradually develops. Obviously in congenital megacolon resection of the nerve-deficient bowel must be complete before intestinal continuity is restored. Biopsy of the bowel at several points and histologic tissue examination provide the limits for resection and indicate the degree of colectomy.

THE SPHINCTERS OF THE RECTUM

The sphincters of the rectum are of practical interest. From a broad anatomic standpoint these are (1) the pelvic sphincters, (2) the internal sphincters, and (3) the external sphincters. The *pelvirectal sphincter* represents a thickening of the circular fibers at the junction between sigmoid and rectum in the region of the angulation of the pelvic colon. This angular constriction has not been conclusively established as an anatomic structure or proved a true physiologic sphincter mechanism. The thickening of the circular fibers, the changes of the mucosa and the narrowing of the bowel in this region are found in at least 80 per cent of cadavers examined in our laboratory. Functionally, the pelvirectal sphincter serves as a restraining barrier for the storage of fecal material in the pelvic colon, and when the latter has passed into the rectum the sphincteric mechanism permits the build-up of the necessary intrarectal pressure for the stimulation of the defecation reflex. From a clinical standpoint the region of the pelvirectal junction seems to be a vulnerable site of origin of several disease processes such as ulcerative colitis and the like.

The *internal sphincter* consists of a thickened ring from the involuntary circular fibers of the rectum at the anorectal junction. The nerve supply of this sphincter is the same as that for the descending colon, the autonomic nervous system. Stimulation of its nerves, however, causes an opposite effect to that obtained from the colon proper. Parasympathetic stimulation results in relaxation, while sympathetic stimulation increases the tonicity of the sphincter. Further, it is interesting to note that the enteric plexus passes beyond the sphincter and may extend into the anus.

The *external sphincter* is composed of voluntary striated muscle which enfolds the anal canal. Its nerves, the inferior hemorrhoidal and perineal nerves, are derived from the sacral plexus. The anterior and posterior sphincterian nerves originate usually from the coccygeal plexus. The sphincter is under voluntary control through the cerebral spinal system. It is in a state of chronic contraction and has no antagonist to oppose its action.

CONGENITAL ANOMALIES OF THE RECTUM

Malformations of the rectum are due to improper growth or arrested development of the hindgut, which in the five-week embryo empties together with the urogenital sinus into the cloaca. The urogenital membrane anteriorly and the anal membrane posteriorly develop from the cloacal membrane as the perineum takes form. Improper closure of the perineal septum leads to fistula formation involving one or more neighboring structures. The rectum proper is derived from entoderm and mesoderm, the former contributing the mucosa, the latter the

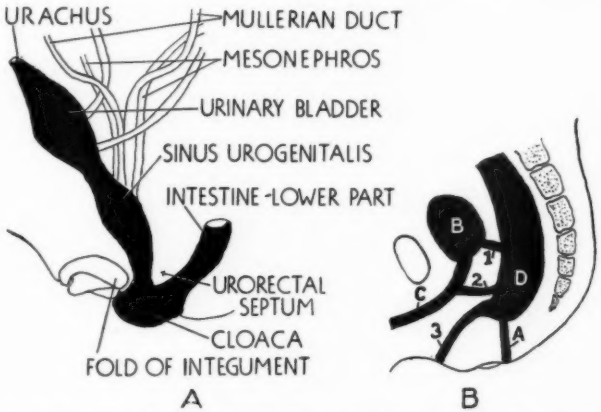

Fig. 8. *A*, Diagram of the hind portion of an embryo showing the common communication of the intestine and urogenital sinus into the cloaca. (After Berman.) *B*, Fistulas associated with failure of closure of the perineal membrane. *1*, Rectovesical fistula; *2*, recto-urethral fistula; *3*, rectoperineal fistula; *A*, congenital narrowing of the anus; *B*, bladder; *C*, urethra; *D*, rectum. (After Berman.)

other constituents of the rectal wall and its supporting structures. Malformations of the rectum can be classified as (1) absence of the rectum (complete); (2) atresia of the distal end of the rectum; (3) occlusion of the rectum; (4) normal rectal development with fistula formation: (*a*) rectovesical fistula, (*b*) recto-urethral fistula, (*c*) recto-vaginal fistula, (*d*) rectoperineal fistula.

It is best to remember that anal malformations commonly exist together with the afore-mentioned rectal deficiencies and that anomalies of the rectum and anus are usually associated with congenital pathology existent simultaneously elsewhere.

For the newest anatomic description of the anal sphincter musculature and its important surgical complications, see J. C. Goligher, A. G. Leacock and J. J. Brossy: Surgical Anatomy of Anal Canal. *Brit. J. Surg.*, 43:51, 1955. (Editor's note.)

132 Parkside Avenue
Brooklyn 26, New York

PROCTOLOGIC EXAMINATION OF THE JUVENILE PATIENT

ROBERT TURELL, M.D.

ANTHONY L. DANZA, M.D.

DAVID W. MOSS, M.D.

EDWARD A. KAMENS, M.D.

HISTORY

A detailed history may usually be obtained from the parent or guardian of the infant or young child. The older child can, in most instances, relate a reliable story. Tactful interrogation by the examiner yields useful additional information. Although a proctologic history seldom suggests a definite diagnosis, it nevertheless is important for orientation.

Appearance of Stool

Any deviation from the normal color and character of the stool is usually noted by the attendants of the juvenile patient; blood-streaked feces particularly impress the parent. A history of change in the character of the feces, such as loose or unusually dry stool, should lead to a comprehensive inventory of the recent foods eaten by the patient.

Protrusion

In the infant and very young child, protrusion through the anal canal is also frequently observed by the parent. However, in older children painless protrusion without accompanying bleeding may escape attention for some time. The protruding mass is usually accurately described by the parent. It is imperative to inquire whether the protruding mass appears only after defecation; whether it is painful,

as manifested by crying of the infant during the course of protrusion; whether it is reduced spontaneously; and whether bleeding accompanies or follows the protrusion.

Pain

The infant usually registers anal pain by crying during defecation or on the introduction of a thermometer into the rectum. The older child not only announces pain to the parent, but also often describes accurately the character of the pain. The intelligent or bright juvenile patient can distinguish between sharp and dull pain, and between intermittent throbbing and constant pain. Sharp pain during and immediately after defecation usually denotes an anal fissure; a throbbing pain points to the presence of perianal or perirectal suppuration; a constant pain is usually indicative of a perianal hematoma, prolapsing rectal mucosa or a protruding polyp.

A detailed note should also be made of pain occurring in the lower half of the abdomen.

Bleeding

Gross bleeding, whether it occurs as streaks on the solid stool or is mixed with either solid or liquid feces, or is passed in its natural liquid or clotted state, whether moderate or profuse, always alarms the parent and the older child and is therefore promptly called to the attention of the clinician. Further inquiry should be made to ascertain whether bleeding occurs before or after defecation, and whether it is bright red or dark brown. Intermittent and painless bleeding, though alarming to the patient during the initial appearance, is, unfortunately, frequently forgotten by the parents in many instances.

Also noteworthy is the fact that while gross loss of blood usually receives proper attention, occult and slow bleeding manifested by a progressive secondary anemia with progressive pallor is frequently overlooked or unrecognized and usually uninvestigated and untreated for a long time. The parents' concern is invariably transferred to the intelligent children who become worried and jittery.

Excretions

The passage of mucus or pus during or independently of defecation in some cases is reported promptly by the parents, while in other instances it is completely ignored or forgotten. For unexplained reasons many parents appear to be secretive about this symptom and do not seek early medical attention.

EXAMINATION

Recto-abdominal Examination

The patient with proctologic symptoms should be accorded a simple visual, palpatory and sometimes auscultatory examination of the abdomen. The lower portion of the abdomen, especially the pelvis in the female patient, should receive a bimanual, recto-abdominal examination. It is surprising how often intra-abdominal or pelvic masses—e.g., appendiceal or adnexal inflammation, small, encapsulated suppurative masses and tumors—are detected on this simple bimanual, recto-abdominal examination. To avoid discomfort and to promote most cooperation on the part of the patient, the gloved examining digit of one hand should be well lubricated and gently passed into the rectum with the patient in the lithotomy position and the knees flexed on the abdomen. The other hand is utilized for the abdominal part of the examination. After a little practice this examination can be performed with dispatch and practically without discomfort to the patient.

Proctoscopic (Noninstrumental) Examination

Inspection. With the patient in the prone, Sims' left lateral, knee-elbow or knee-chest position, the buttocks are separated gently by the examiner or his assistant and the perianal skin is observed for deviations from the normal—e.g., discolorations, eruptions, tumefaction, scars, sinus openings and fissures—and for external invaders—e.g., worms, lice, and so forth. The contour of the anal orifice is observed, and, whenever possible, the patient is asked to strain. This act tends to evert prolapsing mucosa or polyps or to expel excretions, such as mucus, pus or blood.

The pilonidal or sacrococcygeal area, the perineum, the buttocks and the vulva are also inspected at this time.

Palpation. Monodigital. The perianal, perineal and pilonidal areas are gently palpated with the tips of the fingers for evidence of subcutaneous areas of tenderness or invisible masses. A gloved, well lubricated finger, utilizing the little finger for the very young infant and the index finger for patients past one year of age, is then gently insinuated past the anal canal into the rectum with the anterior and posterior aspects of the finger facing the lateral walls of the anus. This maneuver detects anal sphincter spasm, tenderness and masses in the rectum or adjacent organs. At this point the already described bimanual recto-abdominal examination may be performed.

Bidigital. The bidigital examination is then performed by maintaining the index finger in the rectum and placing the thumb of the same hand on the perianal skin. Gentle palpation of the tissues intervening

between the index finger and thumb is then performed in a clockwise or counterclockwise manner until the entire anoperianal circumference is examined. This maneuver will elicit small, circumscribed areas of tenderness pointing to the presence of localized deep areas of inflammation or suppuration, foreign bodies, and so forth. Lesions missed on monodigital examination may become easily discernible on bidigital palpation. It is frequently surprising to observe a quiet, cooperating juvenile (also adult) patient jump or cry out with pain when a localized tender area is palpated while the tissues on either side of the tender area remain nontender.

ENDOSCOPY

Indications

The indications for endoscopy, especially sigmoidoscopy, are (1) bleeding from the anal canal; (2) protrusion from the anal outlet; (3)diarrhea, new or recurrent, which lasts for five or more days; (4) discharge of pus or mucus from the anus; (5) pain in the anoperineal area; (6) prior to the performance of any anorectocolonic operation; (7) for the observation of the progression or regression of intestinal disease; and (8) whenever bacteriologic and histologic studies of colorectal conditions are deemed necessary.

Contraindication

The only real contraindication to endoscopy is the presence of an obstruction which prevents the introduction of the instrument into the anorectocolonic tube.[4]

Instruments

The common observation that the stool passed by children is almost the size of an adult stool supports the view that special, small-sized instruments for endoscopy of juvenile patients are unnecessary except for the newborn or the patient with an incomplete obstruction. The special-sized pediatric instruments afford a reduced range of visibility and are usually the innovation of the inexperienced examiner or are used as a matter of habit, custom or tradition. Adult-sized endoscopes, usually 1.6 cm. (⅝ inch) in diameter, are suitable for juvenile patients except the newborn.[5]

Sigmoidoscope. This instrument is a rigid, hollow tube which is about 25 cm. (10 inches) long and 1.6 cm. (⅝ inch) wide and is available in two types, one with a distally placed (close to the mucosa)

source of illumination, and the other with the source of light placed at its proximal end. The former provides excellent illumination, which, however, frequently becomes dimmed by adherent particles of stool or excretions. These drawbacks are absent in the proximally lighted sigmoidoscopes, the light of which is, however, diffused and consequently is not as bright as that in the distally lighted instrument. Realizing the advantages of each type of illumination, in 1948 I designed a simple sigmoidoscope with perfect proximal and distal illumination which facilitates the examination greatly. Recently this instrument has been further simplified.[6]

The sigmoidoscope has a built-in suction tube which is useful for (1) the removal of fluid material from the bowel, e.g., retained enema fluid, liquid feces, mucus and other excretions; (2) the exhaust of smoke produced by surgical diathermy; and (3) obtaining intestinal washings for culture.

Shorter Instruments. Proctoscopes are usually about half the length of the sigmoidoscope. I use this shorter instrument infrequently, preferring the use of the sigmoidoscope whenever endoscopy is indicated. On the other hand, *anoscopes* are useful for the examination of the anal canal and for treatment of lesions situated about the mucocutaneous junction. The distal end of the anoscope has an oblique rather than round opening, with the source of illumination placed at its proximal end.

Preparation

In over 60 per cent of patients, juvenile or adults, sigmoidoscopy may be performed without preliminary preparation. In the presence of diarrhea it is especially desirable to dispense with preliminary irrigations, since these may aggravate an existing inflammatory process. Preliminary irrigations may wash away blood descending into the sigmoidoscope from areas of the colon beyond the reach of the sigmoidoscope. Preliminary preparation is never omitted whenever anesthetization of the patient is contemplated.

Good preparation for endoscopy consists in the administration of two enemas of 500 to 1000 cc. of plain, lukewarm water or warm normal saline solution. The first enema is usually given 10 to 12 hours before the scheduled time of the examination, the second enema approximately three hours before. Frequently an enema administered in the morning soon after a normal defecation cleanses the terminal portion of the colon and rectum sufficiently well for endoscopy to be carried out at any time within the ensuing eight hours. The simplest

preparation is accomplished by the administration of a colonic irrigation which is to be terminated about an hour before examination. This type of irrigation should be given by a trained nurse. More recently I have used with satisfaction left colon catharsis which consists of an injection into the rectum of about 50 to 100 cc. of solution, each 100 cc. of which contains 16 gm. of sodium biphosphate and 6 gm. of sodium phosphate.

The use of cathartics is neither desirable, necessary nor indicated. It is also unwise to use medicated suppositories, since these coat the rectal mucosa so that small or discrete ulcerations or other lesions may be easily overlooked.

Anesthesia

A good policy is to perform anoscopy and anoproctoscopy on juvenile patients of all ages without the induction of anesthesia, unless a painful anal lesion, such as a fissure, is present. Sigmoidoscopy, on the other hand, is seldom carried out without anesthetization in uncooperative children or those under five years of age. When an anesthetic is given, the examination is preferably performed in a hospital.

Ether, morphine, codeine, barbituric acid derivatives or combinations of these anesthetic agents have been advocated. In the past I noted that morphine, administered in the dosages advised by Paulson,[1] is a safe and satisfactory agent. Recently, however, the drop ether method of inducing anesthesia has been substituted for the administration of morphine on the advice of anesthetists and pediatricians who believe that juvenile patients are extremely sensitive to morphine. Codeine and barbiturates are notoriously unpredictable in their action and constitute poor and undesirable substitutes for either morphine or ether. Again, the administration of these drugs in suppositories is undesirable for reasons already stated.

Older children practically never require anesthesia for sigmoidoscopy. To gain their confidence it is imperative to explain the procedure to them before examination is begun and to assure them repeatedly that this examination, though uncomfortable, is rarely painful. Also, a promise must be made and kept to the effect that should pain occur, the examination will be discontinued forthwith and will be resumed only with the patient's consent after a short wait. It is surprising how amenable these young patients are to such reasoning.

Sedative premedication is usually unnecessary, for this examination is practically painless when skillfully performed. Furthermore, under the influence of sedatives the patient is unable to cooperate and to maintain the special position to be discussed presently. In the presence

of a painful anal lesion, if surgical intervention is decided upon on the basis of history and inspection of the lesion, endoscopy is deferred to be performed in the operating room under anesthesia.

Position

Seven positions useful for the performance of sigmoidoscopy have been described. However, I have found it preferable to perform sigmoidoscopy on juvenile patients, whether anesthetized or not, in the prone, inverted or jackknife position. This position may be effected and maintained by raising the patient's pelvis on one or two pillows. At no time do I use special inverting tables with straps to keep the infant or child "practically immobile."[2]

Older children are examined in the knee-chest or the knee-elbow positions; on rare occasions the left lateral Sims' position is adopted. There is no need for special inverting tables, and the act of lowering the head of the table seems to frighten many of these young patients in spite of assurances.

Technique

At the expense of repetition it should be stated that whenever sigmoidoscopy is performed without anesthesia, the examiner should keep up a conversation at the level of the patient's intelligence. The juvenile, more than the adult patient, needs constant reassurance and should be told what to expect as the examination progresses. Older children frequently take pride in the "vocal participation" in the endoscopic examination.

Monodigital examination should always precede endoscopy. This maneuver prepares the patient for the insertion of the rigid instrument and tends to overcome resistance or spasm of the anal sphincter muscle, thus promoting sphincteric relaxation. A well lubricated (preferably with a water-soluble inert lubricating jelly) and warm endoscope is first gently passed into the rectum. We never use jellies containing anesthetic drugs such as Nupercaine or Pontocaine or the pre-endoscopy application to the anal canal of a mixture containing 4.5 per cent of benzocaine (marketed as Rectalgan), because these anesthetic agents are strong sensitizers of the perianal and anal skin, causing dermatitis and pruritus in children (as well as in adults).

In juvenile patients the endoscope, as in adults, should first be directed somewhat anteriorly in the direction of the umbilicus. The obturator is removed and its tip is inspected for the presence of adherent blood or abnormal excretions. From this point on the endoscope should be

advanced slowly and gently only under direct vision. The lumen of the bowel ahead of the distal end of the endoscope should always be visualized and inspected before the endoscope is advanced. The intestinal lumen is kept open either by atmospheric pressure introduced spontaneously through the open instrument or by air insufflation. Gentle air insufflation helps to overcome spasm and to smooth the mucosal folds, which in turn helps to detect small, otherwise hidden ulcerations, polyps and so forth. Furthermore, in older children, as in adults, air insufflation makes it possible to examine several additional centimeters of the terminal colon beyond the reach of the endoscope. Some examiners do not favor the use of air insufflation largely because of habit and tradition, while other authors have an exaggerated fear of pneumatic rupture of the colon. In an extensive experience in adults, adolescent patients and older children in whom I have performed endoscopy for a distance of 25 cm. or more I have never encountered such mishap or undue discomfort to the patient as a result of the gentle use of air insufflation. Discomfort or mild abdominal cramps in the unanesthetized patient are usually the result of the introduction of the endoscope into the rectosigmoid or sigmoid regardless of whether or not air insufflation is utilized.

In most infants and young children it is possible to insinuate the examining instrument for only about 5 inches (12.5 cm.). At this point a sharp angulation of the sigmoid with looping into the pelvis is encountered which prevents the safe cephalad passage of a rigid endoscope. The technique of endoscopy to this point is easier of performance than in adults because the terminal spine is almost straight by virtue of the fact that the sacrum is practically not curved and its promontory is still undeveloped.

In older children the insinuation of the endoscope for 25 cm. or farther presents the same problem as in adults. The skilled and experienced examiner encounters practically no difficulty in the performance of this endoscopic procedure. On the other hand, the unskilled and inexperienced examiner may and does produce considerable discomfort and pain when examining the terminal portion of the colon cephalad to 4 inches (10 cm.) from the anal verge.

After the introduction of the proctosigmoidoscope to the desired level, it is slowly withdrawn, observing and studying the interior of the bowel. Fluid or feces coming from the bowel ahead of the instrument may be disposed of by means of gentle suction, cotton applicators or, preferably, cotton balls with the aid of an alligator forceps.

The normal mucosa of the older child resembles that of the adult, which, in turn, resembles the normal mucosa of the upper palate. In infants and young children the normal mucosa is considerably redder, but is resistant to trauma.

BIOPSY

All suspicious, accessible intestinal lesions as well as the grossly apparent malignant lesions should be accorded histologic studies prior to the institution of definitive therapy. Specimens, preferably obtained with a cold biopsy forceps, should be taken from the center, the periphery and the mucosa surrounding the base of sessile neoplastic growths and ulcerations. In neoplasms the representative biopsy specimen should include submucosal structures or the basement membrane in order to show the presence or absence of infiltration of the growth. Special care must be exercised in order to avoid perforation of the bowel, since the bowel of juvenile patients is thinner than that of adults. Pedunculated or small sessile growths are best removed in toto at or with the mucosal base for microscopy.

As in adults, mucosal excrescences, no matter how small, should be studied microscopically.

To facilitate the perfomance of biopsy, I have designed a biopsy forceps which embodies several new features.[3] The shaft with its cutting jaws at its distal end has a rotating mechanism of 360 degrees, and a locking device situated near the handle. Thus, the cutting jaws can be made to directly face a lesion situated anywhere in the circumference of the bowel. The cutting jaw is angulated to about 30 degrees and consists of a stationary and a moving jaw, the latter opening and closing sidewise. The stationary blade permits the steadying of the instrument against the lesion, and the sidewise motion of the moving angulated jaw affords better visualization of the fragment of tissue that is in the process of removal. The moving jaw fits into the cutting edge of the stationary jaw, thus producing a shearing action of the jaws. The stationary jaw has two openings to prevent crushing and distortion of the excised specimen, and the bars between these openings prevent loss of the specimen.

To perform biopsy safely it is imperative to secure perfect illumination and to visualize the portion of tissue to be removed. This diagnostic procedure should be performed by one who has had experience and is able to recognize and treat complications such as bleeding. Bleeding may be most safely controlled with ease by means of pressure with cotton balls or by surgical diathermy. Packing of the rectum with gauze is useless and should not be practiced.

A special word about biopsy of adenomas: Some of the suggestions stem from facts deduced from personal experience, while some are based on conjecture. In adults we decidedly advise against fractional biopsy, which implies the removal of bits of tissue from the surface or summit of an adenomatous polyp, because the character of the interior of the body or the base of the lesion is not revealed. We practice "total biopsy," or the removal of the entire polyp at its base in one piece

or in several large segments. Again, this is particularly true in adults in whom adenomas possess real propensity to undergo malignant degeneration. Total biopsy, by procuring the entire lesion both in juvenile and adult patients, has real diagnostic and at the same time therapeutic value. Because of this we feel that the complete removal of an adenoma at its base is the primary and minimal justifiable cause of action; halfway measures are inexcusable. Diagnostic temporizing with an adenoma by piecemeal removal of bits of tissue at various intervals of time amounts to a sort of homicide.

The biopsy may be performed with the air of either cold or electrothermic instruments. However, tiny adenomatous lesions should not be removed by electrothermic means lest most of the removed specimen be rendered useless for microscopy.

The criteria for the early diagnosis of early cancer in adenomas, particularly that of the noninvasive variety, differ enormously with the experience and perhaps the philosophy of the pathologist. It is appreciated that the differentiation between hyperplasia and malignant neoplasia is at times difficult because the dividing line is not always sharp or distinct, and may vary with the pathologist's critical discrimination of interpretation. It also appears that in doubtful cases some pathologists render a diagnosis of malignancy largely for fear of missing one. To date, although we have not observed malignant transformation in adenomas in juvenile patients, we nevertheless maintain an open mind about malignant degeneration in scattered adenomas of children, because theoretically such transformation is a possibility (we are prejudiced against such a possibility only because our data or experience has thus far failed to reveal such an occurrence).

We maintain great reservations about any diagnosis in adenomas that is based on frozen section technique, since the best pathologists can be deceived in both directions. As with other lesions, a benign-appearing adenoma in the frozen section may prove to be malignant in the paraffin section, and vice versa. Mature clinical judgment is therefore of incalculable value in these problems.

In general, a diagnosis of invasive cancer should be made on clinical and pathologic evidence of malignant behavior or invasive characteristics. The diagnosis must be emphatically unequivocal. Equivocation causes or augments fear or cancerphobia on the part of the patient, and frustration and inaction on the part of the clinician.

Papanicolaou Smear Technique

The cytologic examination involving the changes in the nuclear size, shape and staining characteristics of excretions obtained from or below the lesion in the terminal bowel is now in the process of evaluation.

At the present writing, cytologic studies of rectal and colonic smears are reaching a state of perfection to enable them to substantiate the older and established diagnostic methods, namely, endoscopy, biopsy and roentgenoscopy.

OTHER DIAGNOSTIC PROCEDURES

Culture

Bacteriologic and parasitologic examination of feces or excreta is necessary in special instances such as protracted diarrhea.

Material for cultural studies may be obtained by means of an angulated biopsy-like forceps provided with a closed jaw mechanism. With this forceps liquid or solid feces or excreta from the interior of the bowel can be removed easily through an endoscope in a relatively sterile manner. This material is then transferred directly either to culture media and/or onto slides for parasitologic studies or for staining with suitable stains. Another method of obtaining material for cultural studies, particularly intestinal washings at or close to an ulcerated area, is to introduce suitable culture media with the aid of a sterile syringe into the lumen of the bowel through the suction tube incorporated in the shaft of the endoscope. The injected culture medium is recovered by aspiration with the same syringe through the same route. This process is repeated several times, thus obtaining representative intestinal contents or washings.

Feces obtained at endoscopy may be examined for amebic cysts or trophozoites. However, liquid stool passed after the administration of a saline cathartic is preferable. This examination is frequently followed by culturing of the feces for amebas on special media.

The complement fixation test for amebiasis has a place in research, but should not be used to the exclusion of the foregoing tests. Technical difficulties preclude its general use at present.

Chemical Tests

Facilities for the performance of tests for evidence of occult blood should be available in every well run office or clinic and should be performed by all clinicians.

Miscellaneous

Hemoglobin estimations are performed on every patient with proctologic complaints. The cutaneous Frei test, using commercially prepared antigens or sterilized diluted pus obtained from buboes, is especially performed on patients with protracted diarrhea or rectal stric-

tures. The blood sedimentation rate is performed on patients with unexplained pain in the anorectal tissues and whenever suppuration is suspected. Sinking of the red blood cells is accelerated in the presence of early hidden or frank suppurations of the anorectal tissues. The detection of brucellosis, infrequently a cause of diarrhea and/or anemia, demands the use of special agglutination tests.

ROENTGENOGRAPHY

A discussion of this diagnostic procedure is not within the premise of this paper. Suffice it to say here, however, that the broad indications for plain or air and barium double-contrast roentgen studies in juvenile patients are the same as those in adults. It is our practice to use this diagnostic procedure in all cases of questionable diagnosis, diarrhea, bleeding and solitary or multiple polyps, utilizing the newest methods to minimize the formation of fictitious polyps. (See also Roentgenology of the Colon in Infancy and Childhood, by Paul and Juhl, page 27.)

SUMMARY

A discussion of the indications for and technique of a proctologic survey in juvenile patients is presented.

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25 East 83rd Street
New York 28, New York (Dr. Turell)

ROENTGENOLOGY OF THE COLON IN INFANCY AND CHILDHOOD

LESTER W. PAUL, M.D.

JOHN H. JUHL, M.D.

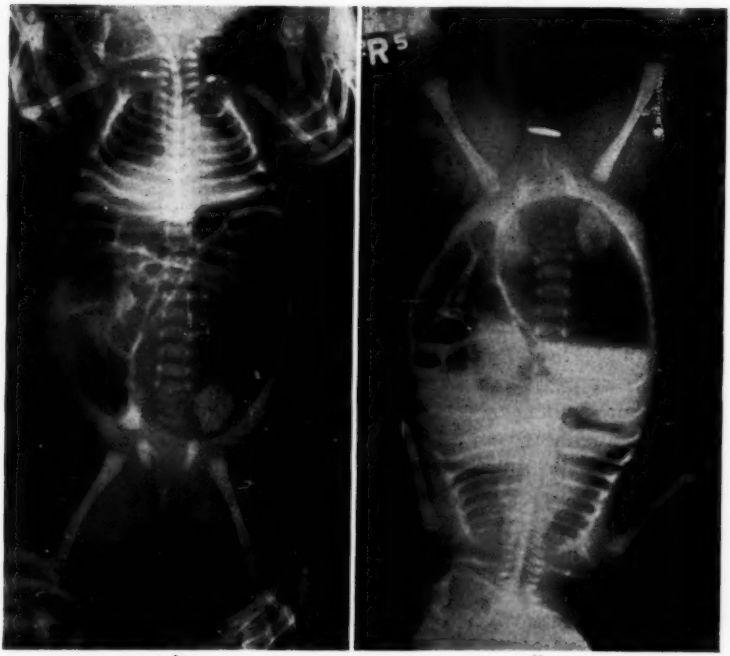
The present discussion is concerned with the examination of the large intestine in infancy and childhood by roentgen methods. The technique of examination and the positive findings in the more common anomalies and diseases of the colon will be considered. Since proctoscopy is the method of choice for the examination of most conditions affecting the rectum, our remarks will be concerned, with certain exceptions, with conditions occurring above the level of proctoscopic vision.

PLAIN FILM EXAMINATION

It is advisable to precede the roentgenologic examination of the colon with preliminary films of the abdomen. The type and number of films will depend, to a large extent, upon the clinical problem presented in the individual case. The simplest and most commonly used projection consists of an anteroposterior view of the abdomen with the patient supine. This will reveal the amount and distribution of gas in the intestinal tract, demonstrate abnormal calcifications if present, and give information about the size of the liver, spleen and kidneys. Abnormal masses may be identified. Swallowed air will be found in the stomach immediately after birth, and within a short time variable amounts of gas can be seen in the small intestine. Normally, gas will be visible in the colon in a matter of a few hours after birth.

From the Department of Radiology, University of Wisconsin Medical School and the University Hospitals, Madison, Wisconsin.

During infancy it is a normal finding to see a considerable amount of gas in the small intestine as well as in the colon. After the age of six months to a year the pattern of gas in the intestinal tract tends to assume that seen in the older child and the adult. Normally, gas is found only in the stomach (swallowed air) and in the colon. Occasionally, short segments of jejunum may be visualized with gas, but



A B

Fig. 9. Anal and rectal atresia. *A*, Supine projection demonstrates gas distention of the intestinal tract with severe dilatation of the left side of the colon. No gas is seen in the rectum. *B*, Anteroposterior projection with the patient suspended by the feet and a metal marker placed on the anal dimple. The distance between the marker and the gas-distended sigmoid indicates that the atresia involves the entire rectum.

any appreciable gas accumulation in the small bowel in the older child has the same significance it has in the adult and must be considered abnormal. During infancy the recumbent position favors the transit of swallowed air into the small bowel and accounts for the common finding of small bowel gas. Gas in the colon, of course, is a normal finding at all ages.

In the presence of megacolon large amounts of inspissated fecal matter intermixed with gas are constantly found. The mottled shadow

produced by these large fecal accumulations produces a characteristic picture of the dilated colon seen in this disease.

In addition to the anteroposterior view of the abdomen, in certain circumstances other views should be obtained. If anal atresia is present, a small metal marker can be fastened to the anal dimple and a film exposed while the patient is held in an inverted position. The gas in the colon will ascend to the highest point, and an idea of the length of the atretic segment can be obtained by noting the distance from the metal marker to the nearest gas accumulation in the rectum or sigmoid. This procedure is of greatest value when the length of the atretic segment is short so that the rectum above it will distend with gas. If the entire rectal lumen or its major portion is obliterated, it may be difficult to be certain just where the atresia ends. Also, failure of gas to enter the rectum with the patient inverted does not always indicate that the entire rectal lumen is obliterated. If the examination is done shortly after birth, not enough gas may be present in the lower colon to be visualized. Accumulations of meconium also may prevent entrance of the gas into the most distal part of the rectum. The sign, therefore, is of greatest value when the atresia is of relatively limited extent and when positive identification of gas within the rectum above the level of the metal marker can be made with certainty (Fig. 9).

Upright films or lateral decubitus views made with the patient lying on one side, the film placed in front of the abdomen and the x-ray beam directed horizontally, are useful if perforation of the rectum or colon is suspected. The presence of free gas beneath the diaphragm in the upright view, or along the lateral margin of the abdominal cavity in the decubitus projection, will establish the diagnosis of pneumoperitoneum. Unless there has been a recent laparotomy or an abdominal paracentesis the presence of free gas in the peritoneal cavity can be taken as a certain indication of perforation of some part of the gastrointestinal tract. It should be noted that after laparotomy air may be visible in the peritoneal cavity for a week or occasionally even longer.

Upright and decubitus views are useful in the diagnosis of obstruction. Only in these positions can fluid levels be observed as the gas in the distended loops rises above the fluid. The presence of fluid levels in the colon is of no significance if enemas have been given prior to the examination. In general, fluid levels in the colon are of less significance than they are in the small intestine in being indicative of obstruction.

BARIUM STUDY OF THE COLON

For most conditions study of the colon by means of a barium enema is the roentgen method of choice. Barium administered orally and fol-

lowed through the intestinal tract may be of some value in the investigation of functional abnormalities and errors of rotation of the colon, but cannot be relied upon for the diagnosis of organic lesions. Barium mixtures should not be given orally if there is any question of an obstructing lesion of the colon because of the tendency for impaction to occur above the lesion. As a rule, therefore, it is wise to begin the roentgenologic investigation of the gastrointestinal tract with a barium enema.

Preparation of the Colon

Preliminary preparation of the infant colon for barium enema study depends to a considerable extent upon the nature of the diagnostic problem. If a search for a source of bleeding is to be made, it is advisable to clean the bowel as thoroughly as possible in order to eliminate gas and fecal matter. The satisfactory demonstration of small polyps can be made only when confusing shadows due to retained fecal matter are not present. In these instances procedures similar to those used in the examination of the adult colon are recommended, including castor oil in suitable doses the evening prior to the examination, the withholding of food on the morning of the examination, and a cleansing enema of warm tap water about an hour before the fluoroscopic study. Less drastic preparation is adequate for most other conditions such as the identification of errors of rotation, partial or complete obstruction and the like. A simple cleansing enema or two will suffice in these cases. In the presence of ulcerative colitis or other inflammatory disease we give a small enema of warm normal saline solution. If a double contrast examination is to be done, it is usually necessary to prepare the colon with castor oil catharsis. This method of study has for its prime purpose the detection of small polyps, and it is essential that fecal matter be eliminated so far as possible.

Barium Mixtures

A mixture of barium sulfate and water with or without a suspending agent is used for barium enema study of the colon. The addition of a small amount of "fluffy" tannic acid powder to the barium-water suspension causes better contraction of the colon and evacuation of the mixture so that the postevacuation film demonstrates the mucosal pattern more clearly. During evacuation of the tannic acid mixture troublesome fecal lumps often are expelled, and the postevacuation film usually shows the colon to better advantage than the filled film. We use the same mixture as for examination of the adult colon. It consists of 300 gm. of barium sulfate powder added to 1000 cc. of

warm tap water. To this is added, if desired, 0.5 gm. of the tannic acid powder to each 100 cc. of mixture. We have found it advantageous to weigh the barium sulfate powder rather than to measure it by volume, since this affords a more accurate control of the density of the mixture and assures a uniform consistency from day to day.

In patients with megacolon it is rarely advisable to attempt to fill the entire colon. Usually there is so much residual feces and gas in the proximal colon that little information is obtained anyway other than the size of the colon above the narrowed segment and, perhaps, some idea of the ability of the colon to contract after being filled with the mixture. It may be difficult to remove the barium from the dilated portion of the bowel, and barium impaction may result. Even the retention of large amounts of water in the dilated atonic colon may be harmful. Since the lesion responsible for congenital megacolon usually is in the rectum or the distal part of the sigmoid, it suffices to fill the left half of the colon in most cases.

Technique of Examination

Fluoroscopy is an essential part of the barium enema study of the colon. The mixture is administered by gravity, the enema can being placed about 2 feet above the level of the table. With most infants and with practically all older children a simple enema tip on the end of the rubber tube suffices. The solution should be allowed to flow slowly in order to avoid undue distention of the rectum, which in turn may precipitate defecation. If difficulty is encountered or expected, a small Foley type of balloon catheter may be used, the balloon being distended with no more than 5 to 10 cc. of air in an infant and 20 cc. in the older child. It is not advisable to use a balloon if ulcerative colitis is known to be present or is suspected, because of the danger of rupture of the rectum.

The flow of mixture through the colon is observed fluoroscopically, and the patient is rotated into various positions to bring the flexures and the loops of sigmoid into profile. Manual palpation with the lead-glove-protected hand is used to separate overlapping loops, to help determine the pliability of walls, and to aid in bringing out small intraluminal defects. After the barium column has reached the cecum the examination is stopped. As a rule reflux into the terminal ileum will occur, and this segment of the small bowel can be examined. Filling of the ileum also serves to locate the ileocecal valve and thus assure that the entire colon has been filled.

A film is exposed with the patient in a supine position, the patient is allowed to evacuate the mixture, and a postevacuation film is obtained. In searching for polyps a double contrast examination can be

done by distending the colon with air after the barium mixture has been expelled. The air is injected under fluoroscopic control, and films in prone, supine and oblique positions are obtained. We have found it advisable as a rule to do the double contrast study as a separate procedure after the routine examination has been done and only if the standard examination has failed to give the necessary information.

THE NORMAL COLON

The length of the normal colon varies considerably in infants and to a somewhat lesser degree in older children. Elongation and redun-

Fig. 10. Postevacuation film of the splenic flexure area of the normal colon showing the fine, crinkled appearance of the mucosal folds.

dancy of the sigmoid are the rule during infancy, but the rest of the colon also may appear unusually redundant. Such elongation tends to diminish as the child grows older. One must use a wide range of variation for the normal before judging that abnormal elongation is present. The width of the normal colonic lumen also varies a good deal when it is fully distended by the enema, particularly if castor oil catharsis has been omitted. Haustral sacculations are less prominent in the infant than in the older child, although they usually can be identified. The more the colon has been distended, the less prominent the haustra become. In the postevacuation state the colon decreases in length as well as in width and the redundant loops diminish considerably. If

tannic acid has been used in the mixture, the colon usually contracts markedly and the mucosa, coated by a thin layer of barium, produces a finely crinkled, "relief" pattern (Fig. 10).

The mobility of segments of the colon also varies widely. The long, redundant sigmoid usually is freely movable because of its long mesentery. In the distended state the flexures rise. The cecum and ascending colon may be unusually mobile because of the presence of a mesentery. Since such mobility, particularly of a very long sigmoid or of the cecum, may lead to volvulus, the condition certainly borders on the pathologic.

CONGENITAL MALFORMATIONS OF THE COLON

Errors of Rotation

The process of rotation of the gastrointestinal tract may be interrupted at any stage of development, giving rise to a number of variations. The foregut and hindgut do not rotate during fetal development and are not involved in these anomalies. The midgut consists of the duodenal loop beyond the first portion, the jejunum, the ileum and the right half of the colon. Growth of the midgut is so rapid that the abdominal cavity cannot contain it, and it enters the umbilical cord in early embryonic development. It rotates 270 degrees in a counterclockwise direction about an axis formed by the superior mesenteric artery. This rotation starts when the prearterial segment enters the abdominal cavity from the umbilical cord in the tenth or eleventh week of development. This segment passes from its anterior position to the right, and then to the left behind the superior mesenteric artery to the left side of the abdomen, where the coils of jejunum fill the left upper quadrant first. The postarterial segment, consisting of the distal ileum, cecum, ascending and proximal transverse colon, then enters the abdominal cavity. It starts from its position posterior to the artery and rotates first to the left and then to the right, anterior to the artery, the cecum finally reaching its position in the right lower abdomen after rotating 270 degrees. The cecum does not actually descend into the right lower quadrant; its position remains fixed in relation to the skeleton. The liver becomes relatively smaller, and the ascending colon grows so that the relation of the hepatic flexure to the cecum is changed without descent. The final stage of the process is the fusion of the mesentery with the posterior abdominal wall. This is particularly important in the prevention of volvulus.

Occasionally a reversal of rotation occurs when the postarterial segment returns to the abdomen first and passes behind the artery from left to right. The transverse colon then lies behind the duodenum; rotation is only 90 degrees in these cases. This anomaly is extremely rare. Complete failure of rotation is also rare; it results in a primitive

arrangement of the bowel, with the jejunum and ileum on the right and the entire colon on the left (Fig. 11). In these cases the distal ileum enters the colon from the right side.

Incomplete rotation (malrotation) is more common than complete failure of rotation. It is always associated with a defect in peritoneal attachment, which is of more clinical importance than the disturbance of rotation. The cecum tends to be abnormally mobile, the attachment of the mesentery is short, and the mesentery itself may be longer than

Fig. 11. Nonrotation of the colon showing the entire colon to the left of the midline. The ileocecal valve is not definitely seen, but the appendix comes off the right side of the cecum (postevacuation film).

normal. In addition there is often failure of fusion between the greater omentum and the mesentery of the transverse colon. These anomalies allow abnormal motion leading to volvulus. There is also a tendency to persistence of abnormal peritoneal bands in these patients with the possibility of compression or kinking at the site of the abnormal band. Many patients with the various types of malrotation experience no difficulty, but others have obstructions of varying degrees which give rise to symptoms which parallel the severity of the obstruction. The tendency is to produce mechanical ileus without obstructing the blood supply.

Occasionally venous return at the mesenteric root is somewhat compromised, particularly when volvulus is present. Bleeding from the

gastrointestinal tract may then be the presenting symptom. Usually this obstruction is not complete, and stools are passed. Many patients may go along for several years with intermittent episodes of cramping pain associated with vomiting until finally the diagnosis is made and the constricting band or volvulus is relieved surgically.

Roentgen Findings. Although films of the abdomen in upright and recumbent positions may be sufficient to make the diagnosis, the

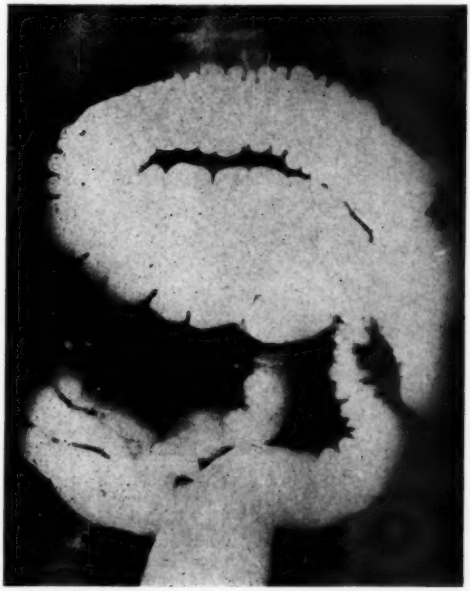


Fig. 12. Abnormal position of the cecum with the ileocecal valve pointing downward and the ileum in the right lower quadrant in relatively normal position. This cecum was unusually mobile as well as in an abnormal position. This is the type of cecum with a long mesentery that may develop into a volvulus.

barium enema is the most important single diagnostic procedure. Barium by mouth is usually contraindicated because of the possibility of producing complete obstruction. It may be given if barium enema findings are inconclusive, however. The important roentgen findings on this examination are the abnormal position of the cecum (Fig. 12) or, if it is in normal position, the determination of abnormal motility of the cecum. Even though the actual obstruction may not be in the colon, abnormal position or motility should lead one to the consideration of extrinsic type of obstruction due to malrotation. It is often not possible and not necessary to determine the exact site of obstruction.

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Volvulus

Failure of normal mesenteric fixation associated with anomalies of rotation may lead to relatively localized volvulus as well as volvulus of the entire small bowel upon its abnormal mesentery. The cecum and the sigmoid are the most frequent sites of volvulus of the colon. Vol-

Fig. 13. Volvulus of the cecum (adult patient). The plain film of the abdomen shows the greatly dilated cecum lying below the transverse colon with its tip to the left of the midline. No gas is seen in the left side of the colon, but a number of gas-distended loops of small bowel are noted, indicating obstruction.

vulus of the cecum is usually found in adults, but may develop in children. It is secondary to an abnormal mesentery which allows increased mobility.

Symptoms depend upon the severity of the obstruction. Pain is usually present and is sharp and crampy and usually localized to the right lower quadrant. Nausea, vomiting and obstipation also occur.

Roentgen findings are usually striking and consist of a large gas-filled loop of bowel, the greatly distended cecum (Fig. 13). It is often seen in the left upper quadrant and sometimes is noted to lie below the transverse colon in the central abdomen. Regardless of its position, the axis of the dilated loop usually points to the right lower quadrant. Distal to the dilated loop there is usually little or no gas, while gas in varying amounts is usually present in the small bowel. When the gas-distended cecum is located in the left upper quadrant, there will be absence of a normal colonic gas pattern in the right lateral abdomen,

a valuable clue in identifying the dilated loop as being a displaced cecum. Barium enema will reveal obstruction at the site of the volvulus with the lumen tapered in a cone-shaped manner almost to a point. In some cases the actual twisted appearance of the mucosa can be made out (Fig. 14). The appearance has been likened to that of the head and beak of a bird of prey. Upright films will usually show a fluid level. The distention of the cecum is out of proportion to the size of the remainder of the colon.

Fig. 14. Volvulus of the sigmoid. This film with barium outlining the rectum and distal sigmoid shows the mucosal relief denoting torsion (arrow) as well as the tapering of the lumen to the area of maximum obstruction and the marked dilatation of the colon above the level of the torsion.

Volvulus of the sigmoid colon is more common than the cecal type. It usually occurs in adults, but occasionally is encountered in children. The symptoms are those of obstruction. Roentgenographic findings are those of a gas-filled loop of large bowel with its axis pointing toward the pelvis. This loop may also contain some fluid which can be visualized in upright views. It may be tremendous in size and extend up to and elevate the diaphragm. There is usually some gas in the remainder of the colon, and there may or may not be dilatation of loops of small intestine. Barium enema will usually confirm the diagnosis. The tapered obstruction is noted in the lower sigmoid with mucosal relief often denoting the torsion (Fig. 14). This finding is typical of volvulus, wherever it may be.

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Duplication of the Colon

Duplication of the colon can occur, but is rare. It can be partial or complete, and the duplicated portion may or may not have its own mesentery. There are no specific symptoms, and the patient may be completely asymptomatic. The diagnosis is difficult because the duplicated portion often does not communicate with the functioning colon. Weber and Dixon[16] reported one case with duplication of the entire large intestine which communicated distally and was outlined by barium.

Imperforate Anus

The diagnosis of imperforate anus is easily made by inspection or digital examination. This anomaly is responsible for about 5 per cent of intestinal obstructions in infancy and is associated with other congenital anomalies in over 50 per cent of cases. Roentgen examination is of value in outlining the distal colon or rectum in order to determine the distance between it and the anal dimple (Fig. 9). The type of surgical correction depends on this distance. A metallic marker is used to locate the site of the anal dimple. Then films are taken in anteroposterior and lateral projections with the infant in the inverted position. The findings can be misleading if gas does not extend into the most distal portion of the colon. Wilson[17] stressed the need for keeping the infant in the head-down position for five to 10 minutes before the films are taken to try to insure that gas will extend into the most distal portion of the colon. Abdominal massage and flexion of the thighs on the abdomen while in the inverted position may also be of help.

Atresia and Stenosis of the Colon

Atresia of the colon is relatively rare. Of a total of 52 cases of atresia of the gastrointestinal tract (excluding atresia of the rectum and anus) reported by Ladd and Gross,[9] only two were in the colon. The atresia can be of two types, either a complete discontinuity or, more rarely, an internal veil or diaphragm which completely blocks the lumen. In the first type, the segments above and below the atresia may be connected by a thin fibrous band, or there may be no connection at all. There may be several atretic areas with isolated blind segments joined by thin fibrous strands which resemble a small string of sausages. Symptoms are those of complete obstruction in the newborn. The presence of meconium does not exclude atresia, but the absence of cornified epithelial cells in the meconium is presumptive evidence that an atresia exists.

Plain films of the abdomen may outline the site of obstruction, since swallowed air will reach the distal colon in three or four hours. Decubitus and inverted positions may be necessary to gain more information than can be obtained with a film in the recumbent position. A barium enema will outline the distal small segment. The colon usually has a narrow lumen if atresia is present above. This should not be mistaken for so-called microcolon, but invariably indicates atresia either of the proximal colon or, more often, of the small intestine.

Congenital stenosis of the colon is also rare. It may vary in degree from a lumen of 2 to 3 mm. to a relatively slight narrowing. Symptoms depend on the severity of the stenosis and are related to the amount of obstruction. Cornified epithelial cells can usually be found in the meconium, since swallowed amniotic fluid can pass through the gastrointestinal tract and contains squamous epithelial cells desquamated from the skin of the fetus. As in atresia, plain films in various positions may outline the lesion if there is gas in the colon above it. If not, a barium enema will outline the site and degree of stenosis.

MEGACOLON

Types

There are three principal types of megacolon—organic, functional and congenital. *Organic megacolon* occurs as a result of obstruction due to tumor, stricture, congenital bands, volvulus, extrinsic pressure, and so forth. It is secondary to these lesions, which can be demonstrated grossly and produce dilatation as a result of mechanical obstruction. *Functional megacolon* is usually due to faulty bowel habits in children and occasionally in mentally disturbed adults. This results in slight to moderate dilatation of the colon to the anus, including the rectum. This condition is not congenital, and the symptoms are mild and rarely produce general debility. In some cases the dilatation may become as great as in Hirschsprung's disease, but it can usually be relieved, at least temporarily, by repeated enemas. The third type is *congenital megacolon*, or Hirschsprung's disease. This disease is characterized by gross dilatation and hypertrophy of the colon without mechanical obstruction. Symptoms are often severe, and death due to the disease is not uncommon. No gross anatomic defect is present, but there is absence of ganglion cells in a segment which results in inability to properly evacuate the colon above it. Constipation is usually present from birth, and the rectum is often free of fecal material in contrast to the functional type, in which the rectum is usually full of feces. The aganglionic segment is normal or decreased in caliber with marked dilatation of the colon proximal to it. The dilatation is usually greater than in the other types of megacolon. This disease is sometimes

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familial and is much more common in males than in females; approximately 80 per cent of reported patients are males.

Congenital Megacolon (Hirschsprung's Disease)

Congenital megacolon was first described by Hirschsprung in 1888. He thought that the dilated bowel was the site of pathology and that it was due to a congenital anomaly. He mentioned the normal caliber and appearance of the rectosigmoid in contrast to the dilated, hypertrophied proximal colon. Since then numerous studies have shown that the narrow or normally sized segment is the actual site of the pathology. Absence of ganglion cells there interferes with coordinated peristaltic action and results in an area of chronic obstruction. Swenson et al.,[13, 14, 15] using a multiple balloon technique, found only small unassociated peristaltic waves in the narrow segment. The dilatation and hypertrophy above the aganglionic segment is a compensatory mechanism.

In addition to the obstruction in the aganglionic segment, the disturbance is enhanced by the loss of the normal defecation reflex, which depends on delivery of fecal material into the rectum. The feces pass into the proximal portion of the aganglionic segment by propulsive action of the hypertrophied colon above it and are then evacuated by voluntary muscle action, which must often be aided by enemas. These patients never have a normal evacuation, and cathartics are ineffective.

The most frequent location of the aganglionic segment is in the lower sigmoid and rectum, but a long segment may be involved, and occasionally there is more than one aganglionic segment. Cases of aganglia involving the entire colon and varying lengths of small bowel have been reported. Keefer and Mokrohisky[8] reported one case with aganglia from the rectum to a point in the ileum 15 cm. proximal to the ileocecal valve. There was perforation of the ileum at the junction of the normal and aganglionic segments. Another of their patients had a long aganglionic segment from the hepatic flexure of the colon to the anus. Swenson found urinary bladder hypotonia in 19 of 20 patients studied, indicating abnormality of parasympathetic innervation to the bladder as well as to the distal colon.

Signs and Symptoms. The symptoms begin at birth or shortly thereafter with constipation, abdominal distention and vomiting. In the newborn, vomiting may be the first symptom. The abdominal distention tends to be progressive; at first it may be relieved by enemas, but eventually it reaches the point at which it is almost impossible to get any relief and acute obstruction occurs, often due to volvulus of the elongated dilated sigmoid. In this event surgical intervention is necessary as an emergency measure. The degree of distention may vary from

time to time, with an increase associated with obstipation. Older children may complain of abdominal cramps. Occasionally the symptoms will be more obscure, with diarrhea at first followed by alternating constipation and diarrhea. Eventually, however, the condition progresses to gross abdominal distention with large fecal masses readily palpable through the thin abdominal wall. These masses are often visible along with the pattern of the dilated colon. Peristaltic waves can also be observed. When this stage is reached, the patient is usually malnourished and anemic. All symptoms and signs are consistent with chronic partial obstruction of the colon. At times administration of tap water enemas may result in a shocklike state, and the patient will be first seen in an emergency room or ward.

Roentgen Findings. A film of the abdomen is often sufficient to make the diagnosis. The greatly dilated colon filled with fecal material which tends to elevate the diaphragm and distend the abdomen is typical of the disease (Fig. 15). The aganglionic segment is not outlined, however, and in order to determine its anatomic site a barium enema is necessary. This examination is not without hazard, since a number of cases of sudden death following tap water enemas have been reported in patients with Hirschsprung's disease. Steinbach et al.[12] reported one death during a barium enema and three other sudden deaths in patients with this disease as well as one patient who recovered from shock which followed an enema. These authors felt that the enemas contributed to the death of the others. Hiatt[7] believes that death is due to water intoxication resulting in a state of low sodium shock. Megacolon has a much larger absorptive surface than the normal colon, and the multiple superficial ulcerations frequently present also contribute to rapid absorption of hypotonic solutions from the colon. The high hydrostatic pressure sometimes used to fill the large thick-walled colon also contributes to rapid absorption. Not all phenomena can be explained on this basis, and such factors as Prostigmine poisoning and mechanical distention of the colon resulting in reflex inhibition of the heart and shock have been considered.

Regardless of the cause, it is important to know that such reactions occur and to make an attempt to prevent them. Steinbach et al. have advanced the following precautions which are easy to carry out and do not lessen the usefulness of the examination: (1)Normal saline solution should be used for the barium enema and for preparatory enemas. (2) An attempt should be made to recover as much of the fluid as possible, using siphoning or suction through a large rectal tube. (3) Avoid using excessive hydrostatic pressure; 2 to 3 feet of water should be sufficient. (4) As soon as the aganglionic segment has been outlined, the procedure should be stopped. It is not necessary to fill the dilated proximal segment. (5) If a shocklike state develops despite these

precautions, the patient should be treated symptomatically with plasma and blood.

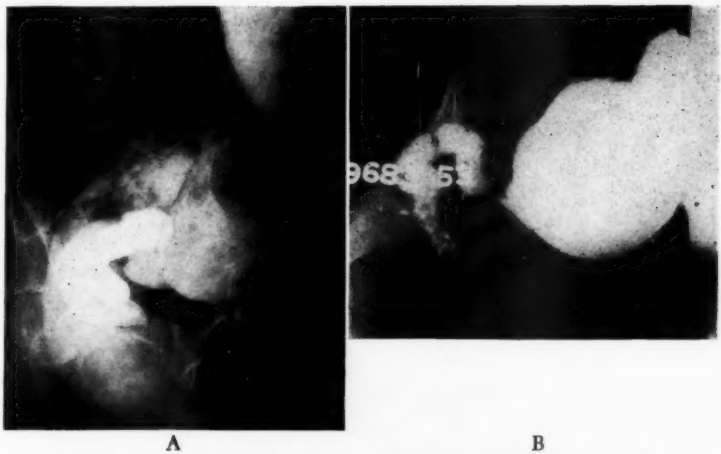
The roentgen diagnosis is dependent upon the demonstration of a relatively narrow segment proximal to which the colon is grossly enlarged (Fig. 16). The narrow segment is usually in the rectosigmoid and may be relatively short. It is therefore necessary to control filling while keeping the head of the barium column under fluoroscopic visual-

Fig. 15. Megacolon. This film of the abdomen of a young boy shows the greatly dilated colon filled with fecal matter and some gas, which is typical of megacolon. The dilated segment fills the pelvis and, if it were full of barium, would obscure the narrowed distal segment in this position.

ization. This segment can usually be readily outlined in the left posterior oblique position, but spot films should be taken in the position in which it is outlined best. Rapid filling without fluoroscopic control can result in failure to see the segment because barium in the dilated colon obscures the narrow area. Once the narrowed segment is outlined and the transition to large caliber is demonstrated, the procedure should be stopped to avoid overdistention and subsequent barium impaction.

In patients with a long aganglionic segment, difficulty may be encountered because of some variation in caliber of this segment. At times it may be larger than normal, but comparison with the greatly dilated proximal colon will show enough difference to indicate that the

aganglionic segment is long and dilated, but that the dilatation above it is much greater. The difference in caliber is therefore the important criterion in these cases. In addition to an increase in diameter, an



A B

Fig. 16. Megacolon. *A*, This oblique view following barium enema shows the narrow rectum and distal sigmoid and the dilated proximal sigmoid typical of this condition. *B*, Megacolon in a 4 year old girl. The spot film in the left posterior oblique position demonstrated the narrow distal rectal segment and the sudden transition to the dilated sigmoid proximal to the obstructing area.

increase in length of the colon proximal to the aganglionic segment is usually present. This may lead to volvulus of varying amounts which adds to the obstruction.

POLYPOID TUMORS OF THE COLON

Polyps of the colon are relatively uncommon lesions during infancy and childhood, but occasionally may be a cause of bleeding from the lower intestinal tract. Since most polyps are small, roentgen examination of the colon must be done carefully. It is essential that the colon be cleaned as completely as possible prior to the barium enema examination in order to eliminate the confusing defects caused by lumps of residual fecal matter (see section on Preparation of the Colon). Fluoroscopic observation during the filling of the colon is necessary. The mixture should be allowed to flow in slowly and the advancing head of the barium column observed. As it passes around a polyp a translucent defect may be noted. This, if the polyp is small, may soon be obscured as the segment of bowel fills more completely. Palpation by the lead-gloved hand over all possible segments of the colon after filling may

bring out the defect of a polyp to better advantage (Fig. 17). Palpation and rotation of the patient serve to unfold overlying loops. "Spot" films can be taken of suspicious areas and are useful in the detection of small polyps. The postevacuation film is an important part of the roentgen study for colonic polyps. If tannic acid is used in the barium mixture, the colon usually empties well and contracts, throwing the mucosa into a fine, crenated relief. The polyp usually retains a thin

Fig. 17. Polyp in the sigmoid colon. Spot film made during fluoroscopy shows the sharply outlined rounded defect of the polyp (arrow) brought out by pressure applied by the lead-gloved hand of the examiner.

coating of the barium mixture and appears as a smooth rounded defect in the normal crinkled mucosal pattern (Fig. 18). If the polyp is pedunculated, as many of them are, the stalk may be seen, and its point of attachment to the bowel wall often causes a slight puckering or indentation of the margin (Fig. 19).

Since the majority of polyps above the level of the rectum occur in the sigmoid and lower part of the descending colon, this area needs to be observed particularly well. If routine barium enema study fails to detect a lesion and a polyp is suspected, usually because of bleeding, a repeat examination for double contrast study of the colon can be done. We have found it better to do this as a separate examination rather than to follow immediately the routine study with a double contrast

examination. Repeat examinations are frequently needed either to confirm the presence of a polyp suspected at the initial examination or because it may be impossible to decide at the first examination whether one is dealing with a polyp or a residual lump of feces.

Fig. 18.

Fig. 19.

Fig. 18. Postevacuation film of splenic flexure area showing a polyp (arrow).
Fig. 19. Pedunculated polyp. Spot films of the colon made during fluoroscopy. The head of the polyp and its long stalk are clearly shown. The point of attachment to the wall of the colon is indicated by the arrow.

The smallest polyp that can be detected roentgenologically is on the order of 0.75 to 1.0 cm. in diameter. Single polyps smaller than this are difficult of detection unless favorably situated along the colonic margin and in an area easily visualized. If multiple, small polyps are easier to recognize because of the multiplicity of defects. Familial

polyposis of the colon is a good example of this. In this disease the surface is studded with innumerable polyps, many of them tiny and no more than a millimeter or two in diameter. Yet because of the number of polyps present the disease can be recognized without too much difficulty, particularly in double contrast films (Fig. 20). The small polyps cause translucent (negative) defects when surrounded by barium

Fig. 20. Multiple familial polyposis of the colon in a young adult. Double contrast film showing numerous tiny polyps throughout the left half of the colon. Most of these are no more than a millimeter or two in diameter.

and positive defects when surrounded by air. The margin of the polyp often has a fine ring contour due to the thin film of barium on its surface. Familial polyposis of the colon is rarely encountered in infancy or early childhood, usually being first detected in early adult life.

OTHER TUMORS OF THE COLON

Carcinoma of the colon is so rare in childhood that it can almost be disregarded as a diagnostic problem. The youngest patient we have seen with a carcinoma of the colon was 16 years of age. Other tumors of the large bowel (leiomyomas, lipomas, and so on) also are infrequent during childhood. These intramural neoplasms resemble, in their roentgen features, sessile polyps with a broad base. The defect is a smoothly rounded one projecting into the colonic lumen. If a mucosal

covering can be demonstrated over the surface of the defect, one can be reasonably certain that the lesion is an intramural tumor, but it usually is impossible to state the histologic type. Such a tumor may be the cause of an intussusception, although this is a more common complication of intramural tumors in adults.

Hodgkin's disease can involve the colon. The defect produced resembles closely that of carcinoma. The lesions may be multiple, a valuable clue to recognition, plus the fact that the patient is in an age period when carcinomas are rarely found.

CHRONIC ULCERATIVE COLITIS

In the majority of cases chronic idiopathic ulcerative colitis begins in the rectum, and the diagnosis can be made on proctoscopic examination. Roentgenologic investigation serves to demonstrate the extent and severity of involvement above the level of proctoscopic vision. Occasionally the disease begins in the sigmoid or even higher in the colon, and roentgen examination is necessary to demonstrate its presence. Infrequently it has its origin in the right half of the colon. Lack of rectal involvement, therefore, by no means excludes chronic ulcerative colitis.

In the early stages of the disease roentgen findings may be meager. Usually the involved segment of the colon is more irritable than the normal as observed under the fluoroscopic screen. Fluoroscopy otherwise may show little abnormality. The films of the filled colon may reveal the presence of haustral sacculations, but these are irregular in width and depth. Often the most information is gained from the postevacuation film. Instead of showing a fine, crinkled appearance, the mucosa tends to be thrown into coarse longitudinal folds (Fig. 21). The thin coating of barium on the mucosa may appear finely stippled because of the presence of innumerable tiny ulcers. In acute fulminating disease the margin of the filled colon may appear rough and shaggy because of the presence of large undermining ulcers.

In more advanced stages of the disease the roentgen pattern becomes fairly typical. Haustral sacculations are absent, the lumen of the colon is narrowed, and the bowel is shortened. The margins of the barium-filled lumen appear finely roughened, and, in the contracted state, the mucosal folds are thickened or completely absent (Fig. 22). The surface coated with barium residue is typically granular. In still later stages the colon assumes the characteristic lead-pipe appearance. It fills rapidly with only a small amount of the mixture. When only a part of the colon is affected, it often is difficult to determine the exact extent of the disease, since the demarcation between the diseased and the normal bowel is not sharp.

When the disease affects the right side of the colon only, the appearance is hardly different than in those cases in which it begins in the distal part of the bowel. With involvement of the cecum, either because of initial right-sided disease or because of generalized inflammation of the entire colon, the ileocecal valve usually is widely patent and rapid reflux occurs through it into the terminal ileum. The ileum often is dilated, although otherwise normal. "Backwash" involvement of the ileum is common in advanced disease. In the late stages of the disease and sometimes earlier, polypoid or pseudopolypoid changes are frequent. It usually is impossible from roentgen evidence to differentiate between inflammatory or pseudopolyps and true adenomatous polyps. The coalescence of small ulcers into larger and deeper ulcerations with undermined edges, leaving islands or tags of inflamed but intact mucosa, causes small, rounded translucent defects in the barium shadow which resemble closely the defects caused by small adenomatous polyps. Since the latter may develop in the colon involved by ulcerative colitis, it is not uncommon to have both pseudopolyps and true polyps in the individual case. Polyposis or pseudopolyposis of the colon secondary to ulcerative colitis presents a typical roentgen appearance. In addition to the usual changes of narrowing, shortening and absence of haustral sacculations, numerous small translucent defects in the shadow will be seen. Those polyps situated along the edges of the bowel and viewed in profile cause the margin to be coarsely irregular (Fig. 23).

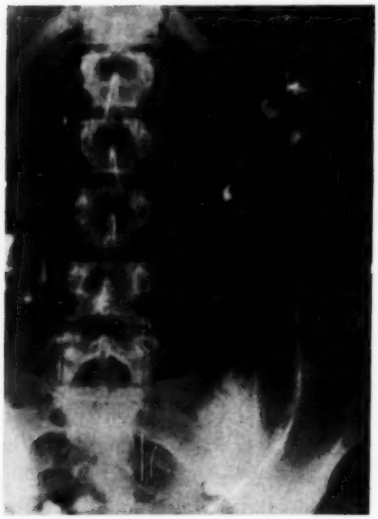


Fig. 21. Postevacuation film in a case of chronic ulcerative colitis. The mucosal folds are thickened and thrown into coarse longitudinal folds. The crinkled appearance is lost. A fine granularity of the surface is present.

Emphasis is directed to the fact that this appearance may be only the result of residual islands of intact mucosa and submucosa which only appear to project into the lumen and thus cause defects resembling true polyps. Thus in the presence of pseudopolyposis improvement in the disease may, in follow-up examinations, show diminution in number or absence of the previous defects. The transition to true polyps cannot be recognized by roentgen examination unless follow-up

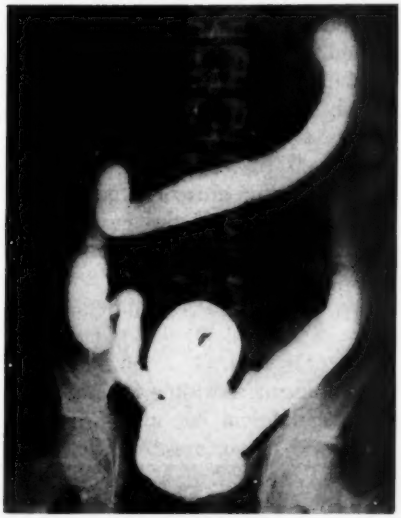


Fig. 22. Chronic ulcerative colitis, advanced stage. Complete loss of haustra, narrowed caliber, widely patent ileocecal valve. Note the relative dilatation of the rectum and distal part of the sigmoid. The disease is of the greatest severity in the right side of the colon, and involvement becomes less intense distally. More often the reverse is true.

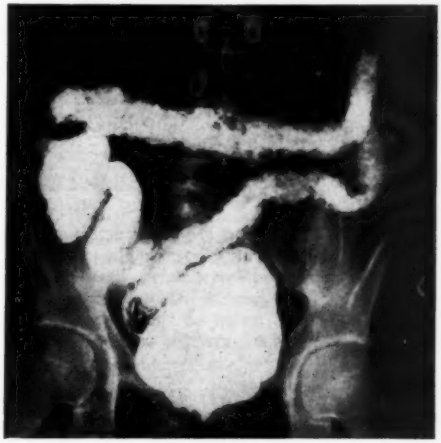


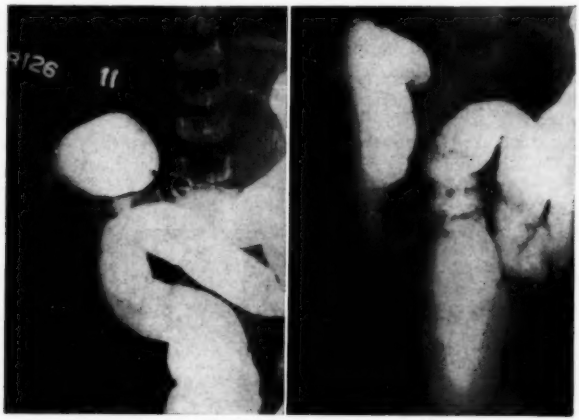
Fig. 23. Polyposis of the colon secondary to chronic ulcerative colitis. The polyps cause numerous translucent defects in the barium shadow.

studies show a constancy of the nodular pattern in the face of clinical improvement in the ulcerative disease.

The differentiation of polyposis secondary to ulcerative colitis and the primary form of familial polyposis of the colon is not difficult. The latter disease is rarely encountered during infancy and childhood. Except for the defects due to the polyps, the colon is normal. The polyps often are so small that they cannot be seen readily in the barium-filled colon or even recognized with certainty in the mucosal pattern of the contracted colon in the postevacuation film. Double contrast studies give the maximum information and often are necessary to demonstrate the lesions or to exclude the disease (Fig. 20).

INTUSSUSCEPTION

The use of a barium enema, not only for confirming or establishing the diagnosis of intussusception, but also for treatment, has gained



A B

Fig. 24. Intussusception. *A*, Typical concave filling defect at the end of the barium column is shown just below the hepatic flexure. The obstructing intussusception, which was first observed in the midtransverse colon, has been reduced to this point. *B*, Further reduction by hydrostatic pressure has been accomplished, but is not complete, and a defect persists in the region of the ileocecal valve. In this instance further reduction could not be obtained, and the patient was operated upon.

increased recognition during recent years. The diagnosis can be made without difficulty. As the barium mixture reaches the site of obstruction the end of the column assumes a concave or cup-shaped form as it surrounds the intussusceptum. Thin ringlike shadows of barium may mark the limit of the shadow, representing barium caught in the haustral crevices as they surround the mass of intussuscepted bowel.

As the hydrostatic pressure of the enema becomes effective it will be noticed that the barium column moves proximally, the concavity of its end being maintained (Fig. 24). If successful, the intussusception may be reduced completely, the entire colon filling, and barium refluxing through the ileocecal valve into the ileum.

Though there has been some objection to the use of this method for the nonoperative reduction of intussusception, the majority opinion seems to be in favor of it, at least as a preliminary measure of treatment. The objections raised have included the danger of rupture of the friable bowel by manipulation during the procedure, the possibility of incomplete reduction being overlooked (persisting ileoileal intussusception after the reduction of the ileocolic phase), and the possibility of missing a lesion which might have been a causative factor, such as a Meckel's diverticulum. We believe that these are not serious objections, provided certain precautions are observed. The enema container should not be elevated more than 3 feet above the level of the table, and only hydrostatic pressure should be used; manual palpation in an attempt to facilitate the reduction should be avoided; free filling of the terminal ileum must be obtained. In addition there should be disappearance of any mass previously palpable and a prompt subsidence of the clinical signs of obstruction. If these precautions are observed, there is little danger in the procedure as shown by published statistics of large series of cases.

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University Hospitals
Madison, Wisconsin

ANESTHESIA FOR PEDIATRIC PROCTOLOGY

BARBARA LIPTON, M.D.

In selecting an anesthetic agent and technique for a pediatric surgical procedure the choice is governed by two considerations: the problems involved in pediatric anesthesia per se, and the difficulties inherent in the particular surgical operation. Any discussion of anesthesia for pediatric proctologic surgery must, therefore, necessarily include first a review of the fundamentals of good anesthesia with special reference to their application in infants and children, and, secondly, the various procedures that come under the heading of pediatric proctology, the anesthesia best suited to each, and the preoperative and postoperative care of these patients.

FUNDAMENTALS OF PEDIATRIC ANESTHESIA

Most of the anesthetic agents and methods commonly used in adults today can be applied to pediatric anesthesia, bearing in mind that the margin of safety in the infant and child is much narrower than in the adult. There are both anatomic and physiologic reasons for the special care required in administering anesthetics to infants and children, and careful attention must be paid to provisions for adequate oxygenation, proper elimination of carbon dioxide and minimal resistance to breathing.

Anatomic and Physiologic Considerations

Many anesthetic fatalities are due to some inefficiency of the respiratory system, and it is this system that is especially vulnerable in the infant and child. Mechanical difficulties may be encountered: the nasal

From the Anesthesia Service, Beth Israel Hospital, New York City.

airway may be blocked partially by adenoid tissue, or beans, beads or other articles which the child has stuffed into his nose. Enlarged tonsils or a wad of gum may obstruct the oropharynx. Obstruction occurs frequently in the young infant when his mouth is closed because, in the absence of teeth, his tongue is pushed against the palate and blocks the oral passage. This can be avoided by use of an oropharyngeal airway or by extending the head with relaxation of the lower jaw.

The infant's trachea is short and has a narrow diameter. The adult trachea is about 10 cm. long with a diameter of 2 to 2.5 cm., that of a newborn about 5.5 cm. long with a diameter of 5 mm., that of a three year old child 8 cm. long with a diameter of 8 mm.[2] The infant's trachea can easily become clogged and the bronchi readily collapsed. Laryngeal edema, thick secretions or aspiration of gastrointestinal contents is therefore a much more severe hazard than in the adult.

In the infant, respiration is carried out almost entirely by the diaphragm while the thoracic wall acts as a fixed point from which the diaphragm can work. Because of the weakness of cartilages and ribs in the newborn there is a certain amount of paradoxical respiration that diminishes the efficiency of respiration. The normal respiratory pattern of the newborn is characterized by rapid, regular respirations interrupted by an occasional deep breath. Sobbing respiration, a two-stage inspiratory effort, is seen in the anesthetized infant and child in instances in which prolonged crying preceded the induction. However, it may occur during anesthesia not preceded by crying. During abdominal operations this may prove troublesome to the surgeon.

The average tidal volume in the infant is 20.5 cc. per minute with an average rate of 28.5 per minute. Rapid rates of 80 to 90 per minute may occur in a normal infant, but are maintained only for one or two minutes. Since one third of the tidal volume represents dead space air, it is obvious that such rapid shallow respirations are inefficient. If such rates persist, they result in exhaustion and inadequate gas exchange. Oxygen consumption in young infants is almost twice that of adults, or 7 cc. per kilogram per minute. Therefore hypoxia may occur with relatively little change in tidal volume. Tidal volume increases with added weight more rapidly than the respiratory rate decreases, resulting in a progressively larger minute volume. The oxygen consumption also increases, reaching a peak of 10 cc. per kilogram per minute between three and five years of age.[7]

In anesthetizing infants one must remember that they have a tendency to ingest gases into the stomach. On inspiration a negative pressure occurs in the stomach, and air is drawn in. If this results in marked gastric dilatation, the infant may have cyanosis, grunting respirations or circulatory collapse. It is also important to note that small infants have extremely active laryngeal reflexes. An endotracheal tube

should never be withdrawn while suction is being applied, since this procedure in the young infant may result in prolonged laryngospasm.

Greater fluctuations of circulatory function occur in the normal infant than in the adult. Blood volume in infants is small: approximately 500 ml. in the newborn and 1000 ml. at the end of the first year.[17] Any blood loss is serious, and it is advisable to replace even the smallest blood loss in children under two years of age.

At birth the systolic blood pressure is 75 to 85 mm. of mercury, the diastolic pressure 30 to 40 mm. During the first two weeks of life the blood pressure rises to 95 to 100/50 to 60 mm. of mercury.[22] The pulse rate of the newborn is 180, decreasing slowly to reach 113 to 127 at one year of age.[3] Any spontaneous or stimulated activity may increase the blood pressure or pulse rate, the latter often becoming so rapid that it is countable only by placing a stethoscope over the precordium. Sinus arrhythmia is a common finding in infants and children, but usually disappears during anesthesia. In older children a tachycardia may be indicative of large doses of atropine or scopolamine, carbon dioxide retention, hyperthermia or shock. Pallor is frequently seen in the anesthetized child. Anoxia, deep ether anesthesia, traction on the intestine or spinal anesthesia may be the causes, and not necessarily blood loss or shock.

Body temperature in the infant and young child varies considerably, and marked hyperthermia or hypothermia may exist. Either one must be readily corrected. Hyperthermia is usually associated with too many drapes, high humidity, closed absorption technique or large doses of belladonna.

No discussion of anatomic considerations in pediatric anesthesia should end without mentioning that the termination of the spinal cord is low in infants, ending opposite the third lumbar vertebra. Spinal tap should, therefore, be done at a lower level. When caudal anesthesia is used in the infant, the possibility of an incomplete sacral arch should be borne in mind. If it were present, this would result in an ineffective caudal anesthesia.

Adequate Oxygenation

One must always be sure that adequate oxygenation is maintained. The child should receive at least 20 per cent oxygen in the inhaled atmosphere. It is not enough merely to administer this adequate oxygen percentage; one must be sure that the airway is patent so that the required oxygen will reach the alveoli and the blood stream. Proper holding of the jaw may be adequate to preserve the airway, but frequently an oropharyngeal airway or endotracheal tube is necessary. Any obstruction of the airway will produce some degree of hypoxia.

Proper Elimination of Carbon Dioxide

An increase in carbon dioxide in the inspired air that is much above the normal can be extremely hazardous for the infant and young child. This hypercarbia can readily result in an increase in respiratory rate, an elevation of blood pressure and a tachycardia. A prominent factor in the production of an increase in carbon dioxide is the amount of rebreathing which a child has to do. It is important to note that a small face mask contains about 50 cc. of dead space. When open drop ether is administered, oxygen should be run under the mask to avoid accumulation of carbon dioxide, and also to provide a higher percentage of oxygen in the inspired gases.

Provision for Minimal Resistance to Breathing

Small children rapidly become fatigued when subjected to the resistance present in the modern anesthesia machine. Their respiratory muscles tire, the respiratory rate increases, and the tidal volume decreases. When the open drop technique is used, there is minimal resistance to breathing. There are times, however, when this technique is not feasible, and others must be used. The closed carbon dioxide absorption system should be used with caution because the amount of resistance present is frequently more than the child can tolerate. A partial rebreathing system has little resistance to overcome, but unless large flows of gases are used (5 to 8 liters per minute), carbon dioxide will build up. The nonrebreathing technique utilizing the valves designed by Digby-Leigh, or Stephen, Slater and Fink is the most suitable for infants. In this method the gases flow into a reservoir bag, and between this and the mask or endotracheal tube are two valves arranged in such a way that the patient can inspire only from the bag, and his expirations are blown off into the atmosphere. In this way it is virtually impossible to accumulate carbon dioxide, and the valves are of such construction that they provide minimal resistance in the circuit.[20]

PREOPERATIVE PREPARATION

Fluid Therapy

Most infants who undergo proctologic surgical procedures require no special fluid and electrolyte therapy unless surgical conditions producing intestinal obstruction are present. These conditions include congenital malformations of the bowel such as malrotation and atresia, which are seen in newborn infants, intussusception and volvulus. All these obstructing conditions may produce severe vomiting which results in loss of water and electrolytes.

It is extremely important that the anesthetist who sees these children preoperatively be aware of the fluid and electrolyte imbalances that occur in various surgical conditions, since correction of them before and during operation may be lifesaving. Children tolerate surgical procedures better and recover more quickly when they are well hydrated and have relatively normal concentrations of body electrolytes.

The principal derangements of fluid and electrolyte equilibrium that may occur in surgical conditions are acidosis, alkalosis, dehydration, ketosis, potassium depletion and chloride deficiency.[13]

Acidosis. Acidosis occurs when intestinal obstruction below the pylorus has been present for more than 18 to 24 hours, and results from loss of alkaline intestinal secretions through vomiting and duodenal suction, from starvation ketosis or from impaired renal function resulting from dehydration and shock. When mild acidosis exists, correction of dehydration and restoration of renal function is sufficient treatment, but in the presence of severe acidosis sixth-molar sodium lactate or sodium bicarbonate solution should be given intravenously. The usual amount of sixth-molar sodium lactate given is 30 ml. per kilogram of body weight.[12, 18]

Alkalosis. When loss of hydrochloric acid from the stomach is relatively greater than loss of alkaline intestinal secretions, alkalosis occurs. This may result because of vomiting or suction drainage. Alkalosis may be corrected through restoration of normal chloride concentrations, and administration of Ringer's solution will return chloride concentrations to normal. Usually, 30 ml. per kilogram of body weight suffices.[12]

Dehydration. Dehydration in surgical conditions is due mainly to lack of fluid intake, vomiting and fever. To correct moderate dehydration 5 per cent glucose in distilled water should be given along with Ringer's solution or isotonic saline. Of the latter solutions, 30 ml. per kilogram of body weight is a sufficient amount until laboratory data become available. When dehydration is so marked that anhydremia and shock are present, the most important aspect of preoperative treatment is to restore normal circulating blood volume and normal kidney function. A whole blood transfusion may be necessary prior to operation. Of course, blood must be administered during operation if much blood loss is anticipated or actually occurs.

Ketosis. Ketosis is produced when food intake ceases and starvation begins. Glycogen stores are then utilized to provide for caloric needs, and when these are exhausted, fat is used to supply energy. Ketone bodies, which are the end products of fat metabolism, are produced in abnormal amounts. Infants should seldom receive anesthesia until the urine is free of ketone bodies. Correction of ketosis can be accomplished by administration of glucose in amounts of 3 to 5 gm. per kilogram of body weight. When rapid correction of dehydration and

ketosis is necessary prior to operation, 5 or 10 per cent glucose in water may be given intravenously.[18]

Potassium Depletion. This results from failure of potassium intake in the presence of continued renal excretion, or from vomiting, diarrhea or diminished reabsorption of gastrointestinal secretions proximal to the site of obstruction. Ten milliliters of potassium chloride–Ringer's solution per kilogram of body weight may be given subcutaneously every six hours after renal function has been restored. However, it is seldom necessary to administer before operation solutions containing high concentrations of potassium, though these may be important in postoperative fluid therapy.

Administration. In conclusion, *intravenous fluids should not be administered at a rate exceeding 20 drops per minute* (60 ml. per hour), and in small infants receiving continuous intravenous fluids the rate should be much slower. The weight and needs of each individual child should be considered and the rate of infusion adjusted accordingly. It should also be remembered that the young infant, until two or three months of age, cannot concentrate urine efficiently, requires relatively more urine to excrete solutes than do adults, and has little reserve capacity for conserving water in pathologic states such as fever and vomiting. Despite this relatively larger obligatory urine excretion, newborns also have a decreased ability to excrete water, and water intoxication may be a hazard during the first three months of life.[14]

Preanesthetic Medication

The impressions that a child receives during his first stay in the hospital usually remain with him for the rest of his life. The child should be admitted to the hospital approximately 24 hours before operation so that he may become acquainted with his surroundings and adequately prepared for the contemplated procedure. This preparation may include fluid therapy, blood transfusion, passage of a nasogastric tube or merely assurance that the infant or child has an empty stomach so that he will not aspirate vomitus in the course of the anesthesia or surgical procedure.

The preoperative visit paid by the anesthetist serves two main functions: first, appraisal of the patient's size and physical status in order to determine the kind and amount of preoperative medication, and, secondly (if the child is old enough), to make friends with him and gain his confidence. Burstein[5] has listed the aims of pediatric preanesthetic preparation to be avoidance of psychic trauma, prevention of mucus secretions during general anesthesia, prevention of reflex hyperactivity, and prevention of respiratory depression from oversedation. We have found a combination of secobarbital and scopolamine, given

intramuscularly one to one and a half hours prior to induction of anesthesia, the most suitable medication for fulfilling the foregoing qualifications. No medication is given infants under six months old. Thereafter the amount of secobarbital given is usually about 10 mg. per year of age, and the amount of scopolamine 0.05 mg. from six months to one year of age, 0.1 mg. from one to three years of age, 0.2 mg. from four to eight years of age, and 0.3 mg. from eight to 14 years of age. The dosage of scopolamine of course should be omitted or reduced in the presence of fever or dehydration. In excessively apprehensive children or those scheduled for spinal or regional analgesia it is recommended that, in addition, morphine (about 1 mg. per year of age) be administered. In these cases the secobarbital may be given two and a half hours before operation, and morphine and scopolamine one hour preoperatively.

Induction of Anesthesia

Even the most judicious preanesthetic medication may be nullified by an unskilled and stormy induction. The induction should be pleasant and devoid of force. This is frequently difficult, but if one can establish rapport with the child, drawing him into conversation about his home or school life, or get him to sing a song or recite a poem, he will often drop off to sleep without fear or struggling. Incidentally, his singing or talking will increase his respiratory exchange and hasten induction.

Agents that are excellent for induction in young children are nitrous oxide and cyclopropane. These gases are heavier than air, and induction is begun by administering them through a rubber tube leading from the machine and held above the patient's face over the bridge of the nose so that he is unaware of the presence of the apparatus. By the time the mask is put on the child's face, he is asleep, or at least he will not recall having his face covered. In older children induction with intravenous Pentothal or Surital is more rapid, but the child may object to having a needle placed in his arm.

Vinethene is a potent yet safe agent widely used for induction. It does, however, have a strong odor, and children may remember having a mask placed over or on their faces. Ethyl ether is an unpleasant agent to use for induction because it necessitates use of a mask and has a pungent and irritating odor. The administration of rectal Pentothal or Avertin is usually not feasible in procedures involving the anus, rectum and colon.

The enormous importance of psychologic preparation and skillful management of children for anesthesia and surgery cannot be overemphasized. Eckenhoff,[9] in a study of 1008 children admitted to the

Hospital of the University of Pennsylvania for otolaryngological operations, found that 17 per cent had personality changes that might be attributed to preanesthetic or anesthetic management.

AGENTS AND TECHNIQUES FOR SURGICAL PROCEDURES IN PEDIATRIC PROCTOLOGY

The various surgical procedures in pediatric proctology for which anesthesia is necessary range from a simple sigmoidoscopy to a laparotomy and bowel resection. The procedure may be an elective one or a pressing emergency, the child may be in excellent general health or severely ill, and the patient may be a premature infant or a 12 year old child. In infants with congenital malformations of the intestine the most pressing problem may be intestinal obstruction. The prone position used in proctosigmoidoscopy presents a problem in maintaining a patent airway. A child admitted for an emergency incision and drainage of an anorectal abscess may have a high fever or a full stomach. Obviously, no one technique or agent can be used in all cases, nor can a formula be devised whereby the type of patient and the surgical operation are added together to result in the proper anesthetic procedure. The anesthetic agent and method used must be custom-tailored for the particular patient, procedure and surgeon. However, a discussion of the many anesthetic agents and techniques now available for pediatric anesthesia may prove edifying to those who are not acquainted with them.

Ether

Diethyl ether is still the most widely used agent for anesthetizing infants and children. It is potent, has a wide margin of safety, is effective in a high concentration of oxygen, and can be administered with simple equipment.[1] In infants and young children it is most frequently given by the open drop method in which the liquid is vaporized via a gauze-covered mask. Oxygen is run under the mask through a catheter to provide a higher concentration in the inspired air and to help reduce the amount of carbon dioxide accumulated under the mask. This method does not, however, allow the anesthetist to assist or control the patient's respirations. When this becomes necessary, other techniques must be used. Older children do best if given ether through an anesthetic machine as is done in adults. The surgical plane of anesthesia is reached more easily and rapidly with this method if the size of the child approaches that of the adult. Ether does have certain disadvantages. It tends to produce a metabolic acidosis, temporarily depress liver function and is a gastric irritant causing nausea and vomiting post-

operatively in many patients. It is also highly explosive in anesthetic mixtures and should not be used in the presence of cautery.[1] Ether provides good muscle relaxation for abdominal procedures and is probably the agent of choice for laparotomies in infants and children such as in the correction of congenital malformations of the rectum and colon.

Cyclopropane

Cyclopropane is a potent agent, excellent for induction in children, but is a powerful respiratory depressant, and surgical relaxation is not obtained until respirations have long been absent. If it is to be used beyond the induction period, a closed absorption system must be employed. Cyclopropane, administered sparingly, can provide good anesthesia for the small and especially the premature infant, using the to-and-fro method and the barest amount of tubing and connectors so as to eliminate as much dead space as possible. If the anesthesia must be deepened, it is best to add ether rather than give the cyclopropane until surgical planes of anesthesia are reached. Cyclopropane may be used with a relaxant drug in older children, but this combination is not advisable in infants. Epinephrine must never be used in the presence of cyclopropane, for in a large percentage of cases this leads to serious arrhythmias and sometimes cardiac arrest. This agent is also explosive in anesthetic concentrations. Cyclopropane may be a good choice in anesthesia for correction of imperforate anus (if the procedure contemplated is simple), for incision and drainage of an anorectal abscess, or simple excision of a fissure or fistula.

Nitrous Oxide, Trichlorethylene, Barbiturates

Nitrous oxide is a most valuable agent in pediatric anesthesia, provided one is aware of its limitations. It is a weak anesthetic, but a potent analgesic agent. It disturbs the body metabolism less than any other agent, and it serves as a powerful analgesic force when used in combination with other agents such as trichlorethylene or intravenous barbiturates. Intravenous barbiturates such as Pentothal or Surital are primarily hypnotics and exert an analgesic effect only by virtue of the depth of hypnosis produced. These hypnotic agents are excellent in combination with the potent analgesic nitrous oxide, and provide adequate anesthesia when a nonexplosive technique is mandatory. The disadvantages in using barbiturates are that they have a tendency to produce respiratory depression and often hypotension.[8]

Trichlorethylene is a liquid which is usually administered along with nitrous oxide and oxygen, and may be substituted for ether in the

vaporizors on the gas machines. It provides good anesthesia for minor procedures, is nonexplosive, and allows the patient to recover quickly and quietly. It does, however, with a small overdose, produce tachypnea and arrhythmias, and therefore must be given cautiously in children. One must remember that this agent should never be used in a carbon dioxide absorption system, for the drug reacts chemically with soda lime to produce dichloracetylene, a compound which may cause permanent neurologic damage or death.[15]

The nonbreathing technique is the best way to administer nitrous oxide or nitrous oxide-trichlorethylene in children. These agents, along with the barbiturates, are good anesthetics for proctosigmoidoscopy, and for other perineal procedures which are not time-consuming, especially when nonexplosive techniques are necessary.

Relaxant Drugs

The use of relaxants in children is avoided by many anesthetists. They are, however, safe, provided one has adequate means of artificial ventilation available.[19] Our British colleagues[4, 21] regularly use them in pediatric anesthesia. Thomas recommends d-tubocurarine hydrochloride in a dosage of 1 mg. per 14 pounds of body weight. Succinylcholine is an excellent short-acting relaxant for children, and 10 to 20 mg. can be given intravenously with impunity. The use of these agents allows the anesthetist to keep the patient in a light plane of anesthesia and still provide adequate muscular relaxation for the surgeon. In poor-risk older children these agents may be lifesaving, but they are not recommended for use in infants and small children because of the danger of producing prolonged apnea in these little people.

Endotracheal Intubation

Preservation of a patent nonobstructed air passage is essential for the well-being of the patient, and endotracheal intubation is an important means of insuring this. This is especially true in procedures for correction of congenital malformations of the intestine, volvulus, intussusception and other conditions in which intestinal obstruction is present. When an anesthetic must be administered in the presence of severe intestinal obstruction, aspiration of gastrointestinal contents is an imminent danger, and, when general anesthesia is contemplated, passage of an endotracheal tube which is well fitted to the glottis is the only means of insuring against this hazard. Newborn infants can usually be intubated awake without anesthesia or with just a topical anesthetic applied to the vocal cords. In older infants and children this method is often not feasible, and they must be intubated rapidly

after the general anesthesia has been administered. It is of utmost importance that a nasogastric tube be in place before the start of anesthesia and that this tube be aspirated at frequent intervals.

The nasogastric tube, however, is still not a safeguard against aspiration, since intestinal contents can rapidly flow into the stomach through a relaxed pyloric sphincter and flood the pharynx too quickly to allow it to be suctioned effectively. This is a frequent occurrence during operation when the surgeon opens the peritoneum and begins to manipulate intestine. Smith,[18] in an excellent paper on indications for endotracheal intubation in pediatric anesthesia, states that among those cases in which endotracheal intubation is mandatory are major operations in the prone position and operations in the presence of intestinal distention or recent feeding. These are three situations which are especially applicable to anesthesia for pediatric proctology, and in which the patient's life may depend on the presence of a patent endotracheal tube.

Regional Analgesia

The use of an endotracheal tube is not necessary, however, if some form of regional analgesia is used. For colonic and anorectal surgery spinal and caudal analgesia may sometimes be the methods of choice. In administering regional analgesia to infants and young children there are a few points which require attention. The child must be well sedated preoperatively, and if he is reasonable enough, the procedure should be explained to him. For the spinal tap a brief period of nitrous oxide, cyclopropane, Vinethene or trichlorethylene keeps the infant quiet and avoids delay. One must remember that in the newborn the cerebrospinal fluid is frequently xanthochromic, is under less pressure, and is less in amount than in older infants. Lumbar puncture is more easily accomplished if the infant is sat up, since this position distends the lumbar sac. Since the spinal cord extends down to the level of the third lumbar verterbra, spinal tap must be done at a lower level. Added difficulties may be encountered owing to the extreme mobility of the infant's spine even if he is given mild inhalation analgesia for the tap.

The older child who is admitted for an emergency procedure in the presence of intestinal distention or a full stomach often does well with spinal analgesia and relatively heavy sedation before and during operation. Stephen and Slater[16] advocate the use in children of 0.1 per cent hypobaric Pontocaine solution according to the Etherington-Wilson formula and give a dose of 0.5 mg. per year of age. They find the method not feasible in children under two years of age. Two per cent procaine solution given in a dosage of 10 mg. per year of age is the

drug of choice if spinal analgesia is contemplated for those under two years of age.

Caudal analgesia in infants and children is excellent for anorectal procedures in which the prone position is to be used. The sacral hiatus is easily palpable in infants and children, making the technique simple and relatively safe; however, the possibility of an incomplete sacral arch must be kept in mind when this method is used. A number 24 gauge needle can be used and 1.5 or 2.0 per cent procaine solution injected through the sacrococcygeal ligament into the caudal canal. This will provide about one hour of analgesia. In older children, as in adults, continuous caudal analgesia by means of a polyethylene catheter placed in the canal is feasible, but should be reserved for procedures which require more than an hour or for which the duration cannot be estimated.

Although Etherington-Wilson[10] reported the use of spinal analgesia in two infants who weighed between 3 and 4 pounds, most anesthetists avoid the use of regional analgesia in small infants, especially for abdominal procedures. In many cases the disadvantages of spinal analgesia outweigh those of endotracheal anesthesia; moreover, the anesthetist is loath to relinquish the excellent control of the patient which the latter provides him.

POSTOPERATIVE MANAGEMENT

In the postoperative period complications and emergencies may arise so unexpectedly that expert nursing care and careful observation are important factors. This is especially true in newborn infants with congenital anomalies of the intestine who have undergone major surgery. Gross[11] has pointed out that many of these infants also have other types of congenital anomalies which may at times be directly responsible for the infant's death. Careful attention to the patient's color, respirations, pulse and heart rate are, therefore, important not only during operation, but also in the recovery period. Oxygen with high humidity should be given to all infants who have had major surgery. A small plastic infant's tent and a flow of 8 or 10 liters of oxygen will provide an atmosphere of 80 to 90 per cent oxygen. The high humidity will help to ameliorate the laryngotracheitis which may follow endotracheal intubation. Proper administration of fluids, including whole blood, is of vital importance, and in older infants and children the administration of small doses of sedatives and occasionally narcotics may be judicious. In premature and newborn infants the use of the Isolette with its controlled temperature and easily regulated oxygen atmosphere is advisable.

A well equipped pediatric floor should have suction apparatus at the

bedside of each postoperative patient, an infant and child bronchoscope and laryngoscope, and an assortment of endotracheal tubes, oropharyngeal airways and suction catheters. Vitamins, antibiotics, cardiac preparations and other drugs should be on hand for administration when necessary. Children who have had general anesthesia should be kept lying in Sims' position with the head lower than the rest of the body until they have completely reacted. This position helps maintain an unobstructed airway by allowing the tongue to fall forward and by preventing the aspiration of secretions. Children who have received regional analgesia should be observed for return of sensation and motor power and for changes in blood pressure. They should not be allowed out of bed for at least six hours after their return from the operating room and may require moderate sedation to prevent restlessness and excessive movement.

Certainly there is no greater need for well trained, intelligent, conscientious doctors and nurses than in the management of a pediatric surgical ward. Not only the physical but also the emotional needs of the small patient must be understood and fulfilled. For many children with congenital anomalies the hospital visit may be one of many, and their experiences during one sojourn may make them either delightful or difficult patients on the next. Above all, one must remember that those who handle children best are those who are genuinely fond of them and whose behavior suggests their affection.

SUMMARY

1. In administering pediatric anesthesia it is necessary to have some knowledge of the differences in anatomy and physiology between the infant or child and the adult. It is essential that the technique used make provision for adequate oxygenation, proper elimination of carbon dioxide and minimal resistance to breathing.

2. Preoperative preparation of the pediatric patient must include psychologic as well as physical evaluation of the child. The kind and amount of preoperative medication should be suited to the needs of the child. Fluid therapy is an important aspect of preoperative management.

3. Techniques used in anesthesia for surgical procedures in pediatric proctology include both general and regional anesthesia. The agents are the same as those for adults. Regional analgesia is most useful for perineal procedures done in the prone position. Endotracheal anesthesia is advocated for laparotomies in infants and young children when intestinal distention is present. Spinal analgesia can safely be given to older children for abdominal procedures in the presence of obstruction or recent feeding.

4. Postoperatively an expert medical staff, a well equipped pediatric floor, and intelligent, understanding management of the infant or child are as necessary to the recovery of the patient as they were before and during operation.

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2 Fifth Avenue
New York 11, New York

IMPERFORATE ANUS AND ASSOCIATED ANOMALIES

WILLIS J. POTTS, M.D.

All parents looking forward to the birth of their baby anticipate that the child will be normal. Fortunately, in the vast majority of instances their wishes are fulfilled. Congenital deformities of every conceivable type may occur, but none is of greater importance than those of the rectum. Present standards of civilization are built on the assumption that sphincters are adequate. Though an inadequate rectum does not shorten life if there is an escape hatch for feces, inadequate rectal control does produce a social burden of tremendous weight.

The percentage of congenital rectal anomalies is not definitely known; they probably occur in some form once in every 3000 to 4000 births, and are frequent enough to command our most rigid attention.

TYPES

Rectal deformities are of many types with all sorts of associated anomalies. *Congenital stenosis* is rare, easily recognized and simply corrected by digital dilatation. The only important point to bear in mind in this condition is the possibility of its being the cause of constipation. Nothing more elaborate than a digital rectal examination is necessary. If there is a stenosis, it will invariably be located in the lower rectum accessible to the finger. It is most desirable to complete necessary dilatations during infancy before memory of pain has developed.

Imperforate anus due to a membranous band is the most satisfactory type of rectal deformity one can have, because the membrane is easily

From the Department of Surgery, Northwestern University Medical School, and the Children's Memorial Hospital, Chicago.

broken and the child will have a normal rectum after a few dilatations. Unfortunately, this simple type also is rare, or we have not had the privilege of seeing it. The only child with a membranous occlusion of the rectum we have seen was cured by a student nurse. The child was admitted with a correct diagnosis of imperforate anus at age 24 hours. Upon admission, according to routine, the nurse proceeded to take the infant's temperature by rectum. She did not know the diagnosis and nonchalantly shoved the thermometer into the baby's rectum. The

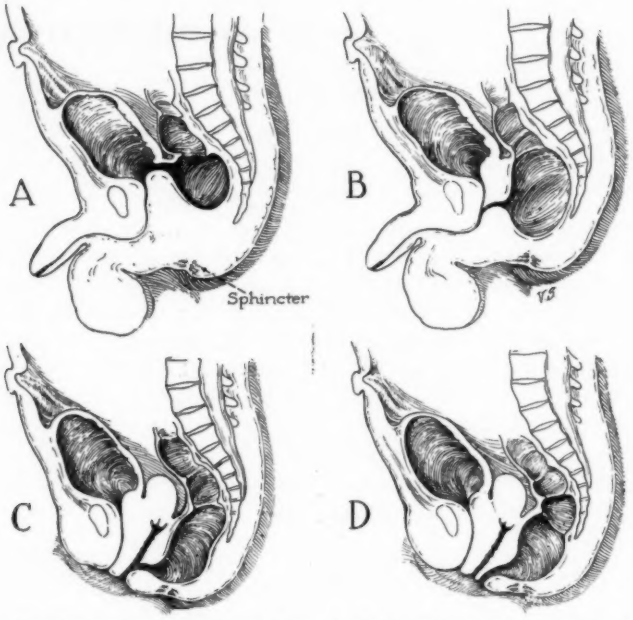

Fig. 25. Types of rectal malformations. *A*, Rectovesical fistula; *B*, recto-urethral fistula; *C*, rectovaginal fistula; *D*, rectoperineal fistula. (Potts, W. J., Riker, W. L., and DeBoer, A., in Annals of Surgery, Vol. 140, published by J. B. Lippincott Co.)

baby promptly passed a large amount of meconium, plus the thermometer, and was cured. (The only thing left for the chagrined surgeon to do was to dilate the rectum a few times.)

The most common, most important and most difficult anomaly to treat is that in which the rectal pouch ends blindly some distance above or outside the normal position of the sphincter. Such a deformity may be and often is associated with a communication with the vagina, urethra, bladder or perineum. In a recent article[1] we reviewed 51 patients who had imperforate anus with such associated anomalies. In this group were 28 girls and 23 boys with the following malformations

(Fig. 25): 22 rectovaginal fistulas; eight recto-urethral fistulas; nine rectoperineal fistulas; 12 rectovesical fistulas. Interestingly enough, in this group no female child had a rectovesical or recto-urethral fistula. For some reason these types occurred only in boys.

DIAGNOSIS

An imperforate anus is easily and promptly diagnosed. If the obstetrician or even the pediatrician should forget to examine the rectum of a newborn child, he will feel a bit apologetic, but no damage will have been done when, in the first 24 hours after birth, the diagnosis manifests itself. However, with associated rectovaginal fistula the diagnosis of imperforate anus may be missed because the infant passes meconium promptly after birth. It is not uncommon to see a child with a rectovaginal fistula unsuspected until at the age of about two years it becomes impossible to toilet train the child. Rectovaginal fistulas usually appear at the fourchette, but may occur anywhere on the posterior wall of the vagina up to the posterior cul-de-sac. Rectoperineal fistulas are recognized at a glance in either sex. In the male child the fistula may be very small and may open along the midline of the scrotum.

The group of cases about to be described are of particular importance. When an infant is admitted without external communications, we routinely gather the available house staff to see the child. After their examination this question is put to them: "What is the most important point to determine next? You see that the child has an imperforate anus. What else do you want to know?" Those who have not previously seen a similar case invariably want an x-ray picture to determine the position of the rectum, or they suggest a physical examination for other abnormalities. Both answers are correct. However, more important at the moment is this: Does the child have a hidden communication between the bladder or uretha and the rectum? To the next question: "How are you going to determine whether there is a communication between the bowel and urinary tract?", the suggestion is apt to be made that the child be catheterized and that contrast medium be injected into the bladder to demonstrate communications. The magic of x-ray pictures for diagnosis still has its hold on students and even a few established physicians.

Actually, the diagnosis of a fistula can be made simply and definitely by examining the urine. A look at the diaper flecked with meconium may give the answer. A receptacle is taped to the perineum and a specimen of urine is collected. Usually particles of meconium can be seen grossly. If there is any question of identification, the specimen is

centrifuged and the sediment examined microscopically. More than one urine examination may be necessary.

Differentiation of a recto-urethral fistula from a rectovesical fistula does require the use of a catheter. If meconium has been identified in the urine, a very fine catheter may be passed into the bladder. If perfectly clear urine is obtained from the bladder, the fistula must be in the urethra.

PHYSICAL AND ROENTGENOLOGIC EXAMINATIONS

Needless to say, a general physical examination is done with special attention to the presence of other congenital abnormalities. It is well known that congenital anomalies are often multiple. Congenital heart disease, atresia of the esophagus and abnormalities of the genitourinary tract are the most commonly associated malformations. Usually the malformations of the rectum take precedence in order of treatment over other anomalies. It is important that every child with any sort of anomaly of the rectum have an intravenous pyelogram. Usually because of distention, the pyelogram will have to be delayed until a later date when the child has well recovered after surgery. Such a pyelogram will be an important part of the record whether normal or abnormal. If abnormal, it will explain later attacks of fever and pus in the urine and will be an important guide to further treatment.

The first 24 hours after birth there is little distention of the abdomen. After 48 hours the abdomen is usually tympanitic and the infant has begun to vomit. Since an imperforate anus is usually seen immediately after birth, these infants are referred to surgery within a few hours after delivery with the anticipation of immediate operative correction. Although operation is an emergency, 24 to 48 hours are often well spent in gathering as much information as possible about the child.

Examination of the anal region will show a ridge of tissue where the rectum should be. Snapping the buttocks or gently pricking the rectal region with a pin will often produce puckering of the sphincter muscle. It is reassuring to know that there is a sphincter muscle, and it is important to note exactly where it is in view of the operation which is to follow.

Roentgenograms are routinely made according to the method of Wangensteen and Rice.[2] A lead marker is placed over the side where the rectum should be, the child is inverted, and anteroposterior and lateral views are taken. During the first 24 hours after birth it is not unusual to find that air has not yet found its way to the end of the colon. By the second day of life the position of the end of the blind pouch is demonstrable.

OPERATION

When operation should be done and whether it should be done in stages are still matters of dispute. Obviously an obstruction must be relieved. We have eliminated the preliminary colostomy. If the child has a rectoperineal or rectovaginal fistula which is large enough for adequate discharge of feces or can easily be dilated to sufficient size, operation may well be delayed until the child is four to six months old, but not longer. By that time the structures have grown somewhat and operative repair is therefore easier; we feel rather strongly that to do a colostomy and leave it until the child is a few years old is a mistake. Eventually the perineal repair becomes necessary, and nothing will have been gained by waiting. Postoperative dilatations are almost invariably necessary, and to do these on a child above two years of age is a nightmare to both doctor and child. This painful procedure should be completed before the child is old enough to retain memory of pain. Furthermore, nothing is gained by waiting. The sooner the rectal mechanism is restored to as near normal as possible, the sooner the infant can begin its subconscious education.

Neither do we believe in preliminary colostomies for infants with imperforate anus complicated by communications with the bladder or urethra, and for the same reasons.

Although this article is written primarily for pediatricians, it seems altogether appropriate that they glance over these words and at least look at the pictures to familiarize themselves with the methods pediatric surgeons are using to correct these deformities. Details of technique will be omitted. Just enough will be given to furnish a general idea of what we are trying to do.

A rectum is not just a hole between the buttocks. We have seen patients in whom a stab wound was made in the perineum with a knife and even with a cautery. The last state of such infants is a sad one. A stricture of iron-hard fibrous tissue forms and is almost impossible to dilate. If the infant could talk, it would plead most entreatingly that the greatest care be exercised with its vital evacuating mechanism.

Preoperative care is rather routine. If the abdomen is distended, transnasal suction is necessary even though the child has not vomited. Problems of fluid balance are minimal the first two days of life. Blood is always made available for all operations of any magnitude.

For this operation as for all operations during infancy, except open thorax procedures, no anesthesia to date is superior to or safer than open drop ether with a tube of oxygen fed beneath the mask.

A polyethylene tube is inserted into some vein for the administration of blood during the operation and for fluids after operation.

Operative Procedures

A one-stage abdominoperineal operation is now done routinely on all infants with rectovesical or rectoperineal fistulas. Only if the child is in very bad condition is a preliminary colostomy performed. Colostomy as a preliminary procedure in the average infant in this group has been abandoned since the days of adequate blood transfusions and effective antibiotics. A colostomy is unsatisfactory because the distal end of the bowel is constantly contaminated and feeds infection into the urinary tract.

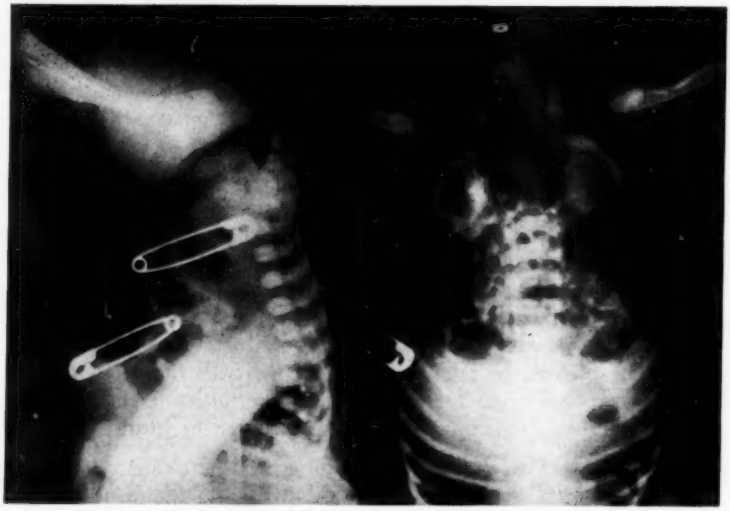

Fig. 26. Roentgenograms of child in inverted position. The lead marker shows the point where the rectum should be. Whenever the gas bubble in the terminal bowel lies this high in the abdomen, the chances of communication with the urinary tract are great.

A catheter is introduced into the bladder. With the child in proper position on the operating table, a short longitudinal incision is made through the skin over the site of the rectum, and the previously demonstrated and marked sphincter muscle is carefully spread with a hemostat until it is large enough to receive the bowel at a later stage of the operation.

The abdomen is then opened, the colon deflated if greatly distended, and the communication between the bowel and the bladder or urethra closed with a few appropriate sutures. Now a long forceps is introduced through the opening in the perineum into the abdomen where the bowel is grasped and brought down through the sphincter. Here the open bowel end is sutured to the edges of the skin around the sphincter. The abdomen is closed and the operation is complete. The most important step in the procedure is to bring down the bowel to the skin where it is sutured *without tension*. If the mucosa of the bowel pulls away from the skin, there will be a raw surface without mucosal covering, and at that point a fibrous stricture will develop.

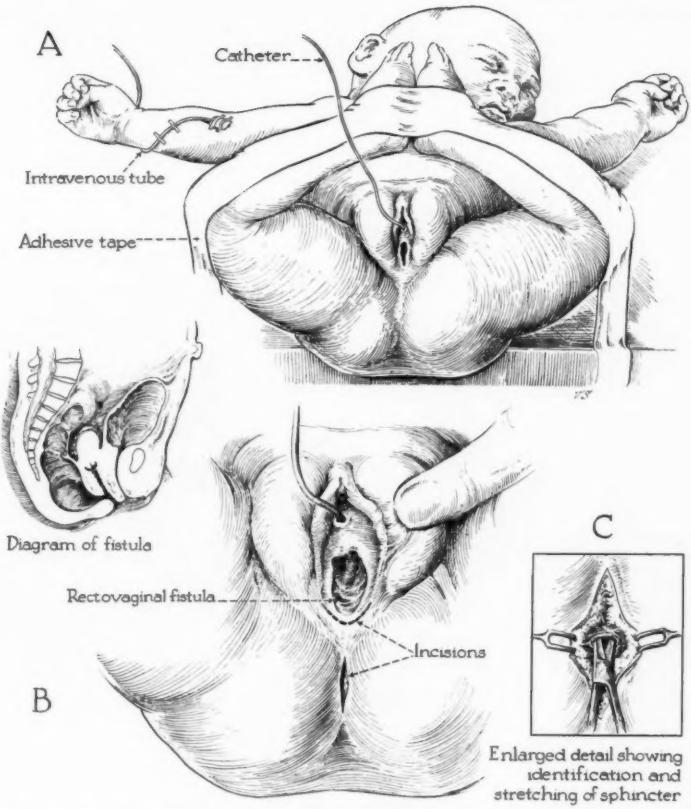

Fig. 27. A, The child is on the operating table, and its legs are kept out of the way by strapping them across the abdomen. In every operation for imperforate anus a catheter is placed in the bladder. B, Two incisions are made rather than one because healing is better. C, The sphincter muscle is carefully spread. (Potts, W. J., Riker, W. L., and DeBoer, A., in Annals of Surgery, Vol. 140, published by J. B. Lippincott Co.)

Rectovaginal and rectoperineal fistulas may be operated upon during the neonatal period, or operation may be delayed for a few months, provided an adequate opening is maintained for discharge of feces. It is not wise to delay operation too long, because as soon as the infant begins taking solid foods there is increased chance of fecal impaction and trouble.

The operation for rectovaginal fistula is illustrated in Figures 26 and 27. One step in this operation which we have developed and which in recent years has yielded rather good results consists in placing the bowel *behind* the perineal raphe. The legends call attention to some of the points of greatest interest.

POSTOPERATIVE CARE

Do not skip the next few paragraphs. Often the pediatrician has to play an important part in the follow-up care of these children.

The infant usually remains in the hospital for 10 to 12 days after an uneventful operation. Although the rectum has been sewn in place and has healed properly, there will be a tendency for it to constrict. Prac-

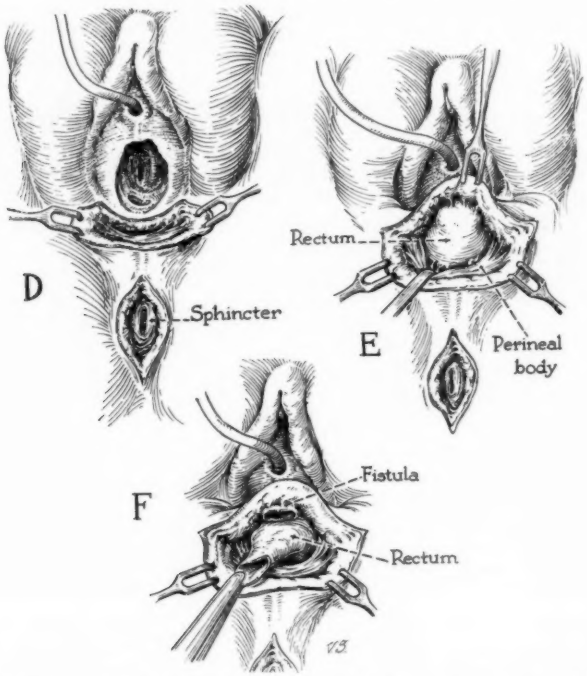

Fig. 27 (*continued*). *D* and *E*, The rectum and its terminal fistula are carefully and adequately dissected from the vagina and *from the perineal body*, which is carefully kept intact. *F*, Rectum freed from surrounding structures. (Potts, W. J., Riker, W. L., and DeBoer, A., in Annals of Surgery, Vol. 140, published by J. B. Lippincott Co.)

tically every child requires dilatation of the rectum after operation. Dilatations are begun carefully about the tenth day and are continued by the mother at home, but require supervision. Many patients who live beyond the suburbs must be helped by their pediatrician or family doctor. The mother is instructed at home to use her little finger protected with an ordinary finger cot. It is important that the patient be checked at least weekly. Diet must be controlled so that the movements are soft, not liquid, or the buttocks will become excoriated; not

hard, or impactions will develop. The latter problem is a real one. At every visit the abdomen is examined for the presence of fecal impactions. Mineral oil by mouth is avoided because it tends to leak through the rectum with liquid feces which make the buttocks sore. Dilatations are performed every other day for a couple of weeks, then biweekly

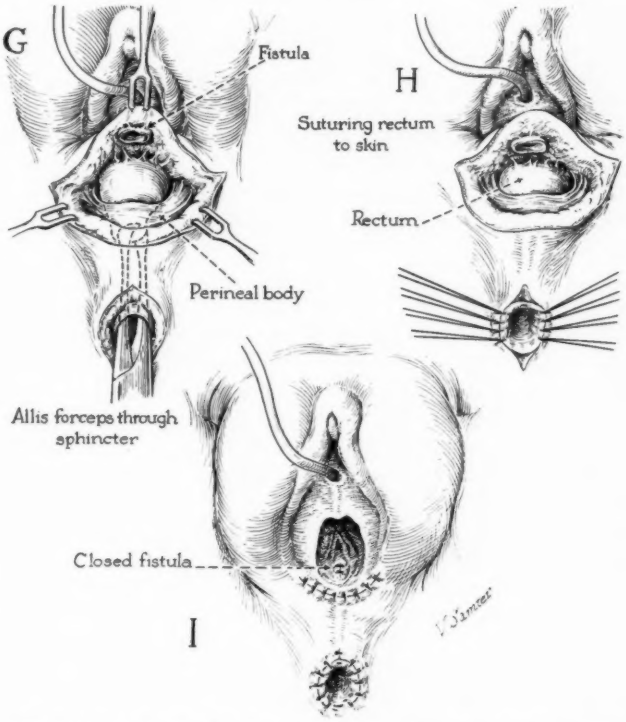

Fig. 27 (*continued*). *G*, This is a new and important step in the operation. An Allis forceps is introduced through the sphincter, and the bowel is drawn down *behind* the perineal raphe, where it should lie normally. *H*, The bowel is now inside the sphincter, where it is sutured to the skin. *I*, The operation is completed. (Potts, W. J., Riker, W. L., and DeBoer, A., in Annals of Surgery, Vol. 140, published by J. B. Lippincott Co.)

until the mother's first finger will go in over the second joint. When this can be done without resistance, an occasional dilatation is in order to be sure that the rectum is remaining open.

One of the most disturbing postoperative problems is that of fecal impaction. Remember that the child was born without the distal part of its rectum and its nerves which form a very important reflex arc. The rectal pouch and its nerve supply are important in normal evacua-

tion. When the rectum is filled, the nerve reflex normally gives the sensation that the bowel should be evacuated. When the distal portion of the rectum is absent and the lower portion of the rectosigmoid is brought down, it is obvious that this important reflex is lost. Instead of having normal spontaneous reflex bowel evacuation, the child must learn to move its bowel. This process literally requires years, and during this time danger of impaction is always present. The process of re-educating the growing child into proper bowel habits is a long, tedious and often discouraging process. Many of these children cannot be toilet trained as are other children. If there is a tendency towards repeated impaction, we have the mother use a suppository or give a small enema each day. These artificial methods of stimulating bowel evacuation not only prevent impaction, but also tend to teach the child subconsciously the method of bowel evacuation.

RESULTS

In spite of all the care, skill, diligence and judgment, some of these children do not have good results. In a follow-up study over a period of years we have found that the children who have had a long missing segment of the rectum with a rectovesical or recto-urethral fistula, whose bowel has been brought down satisfactorily, tend to have an open rectum. They slowly develop some control with the aid of the levator muscles and buttocks.

After proper size of the rectum has been established these children are kept on a constipating diet. Occasional accidents are thus less distressing, and the formed stool helps in the process of bowel education. One cannot promise these parents that their child will have adequate sphincter control. So long as they are constipated they do well. Diarrhea is a nemesis.

The children who have had rectovaginal or rectoperineal fistula repair by placing the bowel behind the perineal raphe where it normally lies have far better results. In fact, some of these children have normal rectums with full control.

CONCLUSIONS

Congenital malformations of the rectum present many surgical and postoperative problems. All things considered, the most important muscles of the body are the sphincters. It is the duty of the surgeon and all those who are engaged in the care of these unfortunate children to make it possible for them to live nobly as well as long.

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707 Fullerton Avenue
Chicago 14, Illinois

OBSTRUCTIVE MALFORMATIONS OF THE COLON

DONALD A. DAVIS, M.D.

Intestinal obstruction in the newborn and young infant carries with it a high rate of morbidity and mortality. During the past 15 years this rate of morbidity and mortality has decreased considerably. This decrease is due primarily to two factors: first, earlier diagnosis and, secondly, improvement in surgical management. There is still room for much more improvement in both quarters.

Intestinal obstruction in the infant is almost invariably due to some congenital malformation along the gastrointestinal tract. The malformations encountered are atresia and stenosis, duplication, malrotation and congenital aganglionosis (Hirschsprung's disease).

ATRESIA AND STENOSIS

These two malformations may be considered together, since they are really only degrees of the same defect. Some writers distinguish between them; others make no distinction. The word "stenosis" means a narrowing. Stenosis of the lumen of the bowel may be primary or secondary and is therefore a poor term to use when referring to congenital primary narrowing. The word "atresia" is derived from the Greek word *atretos*, meaning not perforated. In medicine atresia has, through common usage, become the term denoting complete or partial obstruction due to a congenital malformation in the bowel wall. Evans[5] has suggested the terms "complete" and "partial" atresias and feels that they will lead to less confusion on this subject.

From the Department of Surgery, New York University Post-Graduate Medical School, New York City.

Incidence

The literature leads one to believe that atresias of the intestinal tract occur once in every 20,000 births. However, these figures are compiled in the main from hospital cases, and only a small percentage of births takes place in hospitals. Evans,[5] after a careful and extensive review of the entire world literature on the subject, feels that a corrected figure would be something more like one in every 1500 births. Hence there must be several thousand infants succumbing to intestinal obstruction in the United States each year. Female infants are equally affected. There is no known familial incidence. The distribution along the intestine is approximately as follows: the ileum is by far the most commonly involved; the duodenum is next most common; the jejunum is third; the region of the ileocecal valve and the colon is a poor fourth.

Etiology

There are many theories in the literature to explain these malformations. The most widely accepted is as follows: The intestinal tract before the fifth fetal week is a straight, open tube. Between the fifth and tenth fetal weeks the epithelium proliferates and forms epithelial concrescences. This is termed the solid stage and is the stage of elongation of the bowel. The solid epithelial mass then vacuolates. The vacuoles coalesce, and finally, by the twelfth fetal week, the continuous lumen has re-formed. Atresias, according to this theory, occur during the vacuolation stage; i.e., some region or regions fail to vacuolate, leaving a lumenless portion of bowel. Many observers disagree with this theory, but until a better one is evolved this one will have to stand.

Diagnosis

Atresia is the most common cause of intestinal obstruction in the newborn and in the infant in the first few weeks of life.

Since we are concerned here only with the atresias of the colon, the ratio of obstruction in this period of life is necessarily greatly altered. By far the atresias of the colon are outnumbered by the atresias of the small intestine and duodenum.

The diagnosis should be made in the first 24 to 48 hours of life. Examination at birth reveals a normal anal canal. However, no meconium is passed. There may be a small amount of mucoid material passed, but it is scanty and light in color. This material may be examined for vernix cells (vernix caseosa, which is desquamated epithelium from the fetus' skin swallowed in utero by the fetus). If no vernix cells are found, it must be assumed that there is obstruction somewhere along the intestinal tract. In colonic atresia the abdomen becomes

distended. Extreme distention does not occur until 24 to 36 hours have passed. The intestinal pattern with peristaltic waves may be seen over the abdomen. Vomiting is not an early symptom with large bowel obstruction. It usually is not seen until the second or third day of life. Dehydration from loss of fluids into the distended bowel and from vomiting is rapid in infants with resultant elevation of temperature to 103 to 104° F. and obvious toxicity.

X-ray examination of the abdomen is of great assistance in revealing the level of obstruction. It is rarely necessary to use contrast medium. Barium should never be used by mouth. Barium colon enema is occasionally helpful, but certainly adds time to the work-up and distress to an already sick infant. The flat film and upright film of the abdomen in the vast majority of cases give one all the information needed. In colon obstruction the gas pattern outlines the colon quite well up to the point of obstruction. There are fluid levels on the upright film. There is one variation from this pattern, and this is seen in meconium ileus, in which there are usually no fluid levels. The terminal ileum and first portion of the colon appear to contain a coarse granulated material. Zimmer[21] believes that the lack of fluid levels is due to the filling of the bowel with thick mucoid or inspissated meconium.

Treatment

There is only one treatment for intestinal obstruction in the infant, and that is surgery as soon as the patient is properly prepared. Preparation of the patient means replacement of lost fluids with as near complete hydration as possible; the introduction into the stomach of a number 10 French catheter or polyethylene tube; lavage and gastric suction; oxygen administration; and reduction of temperature below 102° F. maximum before operation.

It is not within the scope of this paper to describe surgical techniques. Some surgeons still feel safer with the Mikulicz type of exteriorization of the two blind ends. The author and some other surgeons feel that a primary anastomosis is possible in most instances, thus avoiding the dangers of an ileostomy or high colostomy. There is the added advantage of one operation instead of two.

Postoperative Management

The postoperative care of these infants is as important as the operation itself. Fluid and electrolyte balance must be watched constantly. Blood must be administered promptly when needed. The stomach should be lavaged and deflated several times daily. Feedings should not be started until definite proof of free passage is obtained. Chemotherapy should be introduced only when indicated.

Summary

In summary it should be emphasized that there are several important steps to take to reduce the high mortality rate in complete and partial atresias of the bowel. These are (1) early and accurate diagnosis; (2) adequate preoperative preparation of the patient; (3) early operation done by a surgeon experienced in infant surgery; (4) watchful, continuous postoperative care.

ROTATION AND MALROTATION OF THE GUT

This subject is discussed not because the colon commonly becomes obstructed, owing to rotation defects, but rather because the cecum and ascending colon are involved in many of the midgut obstructions and the other pathologic conditions which are due to malrotation.

Mechanism of Rotation

In order to understand the mechanisms of the pathologic conditions due to malrotation of the gut, one must have some understanding of the mechanism of rotation itself.

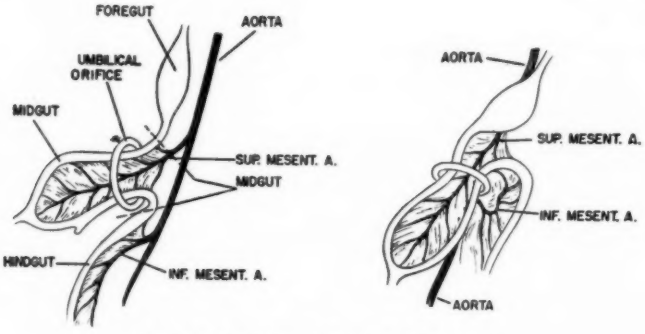

Fig. 28. Rotation of the gut, first stage.

The rotation of the gut has been divided into three stages by Frazer and Robbins.[8] Prior to this, embryologists divided the primitive intestinal tract into three parts: the foregut (mouth to duodenojejunal junction), the midgut (duodenojejunal junction to the midtransverse colon), the hindgut (midtransverse colon to the anus). The intestinal tract in the early weeks of embryo life is a straight tube with a common dorsal mesentery. This tube grows much faster than the celomic cavity and therefore seeks more room. This is found in the umbilical cord. Rotation of the gut begins during its sojourn within the umbilical cord.

First Stage. The first stage begins about the eighth week of fetal life. Normally the midgut is all that is present within the cord. This portion of gut rotates 90 degrees in a counterclockwise direction upon the superior mesenteric artery as its axis (Fig. 28).

Arrest during the first stage of rotation results in an *omphalocele.* Usually the omphalocele contains midgut only. However, at times most of the colon, and portions of the liver, spleen, stomach and even kidney, may be found in the omphalocele. The covering of an omphalocele is the translucent amniotic membrane.

Surgical intervention should be instituted immediately, since the membrane has no blood supply and begins degeneration promptly.

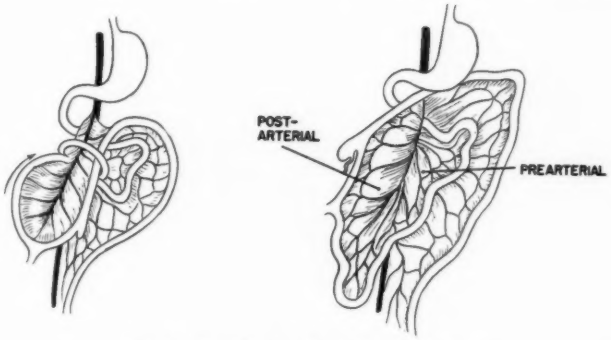

Fig. 29. Rotation of the gut, second stage.

Second Stage. The second stage of rotation occurs about the tenth week of fetal life. It is the most important and most complex of the three stages. During this stage the portion of the gut lying within the umbilical cord returns to the abdomen and in the process of doing so rotates another 180 degrees counterclockwise, making a total of 270 degrees' rotation. The jejunum returns first, and then in orderly fashion the ileum, cecum, ascending colon and right half of the transverse colon return to the abdomen. The small intestine collects in the left side of the cavity and in doing so deflects the hindgut to its normal position along the left gutter. The cecum and ascending colon are then deflected toward the right gutter as they enter the abdomen. This completes the 180-degree rotation of the second stage (Fig. 29).

PATHOLOGIC CONDITIONS. There are several pathologic conditions derived from defects of rotation during this stage. The midgut may return to the abdomen without rotation. In this instance the small intestine rests in the right side of the abdomen and the colon in the left side. Pathologic conditions which may arise from nonrotation are left-sided appendicitis, volvulus of the cecum and other portions of the colon,

obstruction of small and large bowel from congenital adhesive bands.

Another result of defective rotation in the second stage is *volvulus* of the midgut about the root of the mesentery. This condition takes place either in utero or immediately after birth. In this condition the patient may have symptoms of partial or complete obstruction due to twisting of small and large bowel about the mesenteric root. Further, there may be partial or complete occlusion of the superior mesenteric vessels with resultant infarction of portions or all of the midgut. The symptoms resulting from volvulus are those of high duodenal occlusion and/or the toxic symptoms of vascular accident in the bowel mesentery.

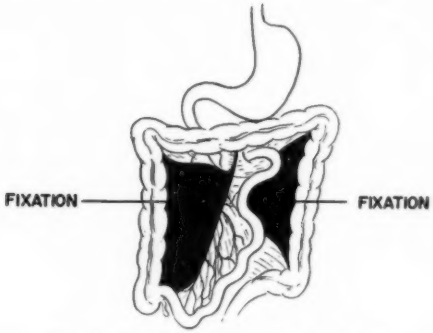

Fig. 30. Rotation of the gut, third stage.

Reversed rotation or clockwise rotation is a third defect seen in second stage rotation. In this type of malrotation the duodenum comes to lie anterior to the superior mesenteric artery and the transverse colon behind it. If normal fixation of the mesentery takes place, the colon becomes trapped behind the mesentery and may readily result in obstruction of the transverse colon at the point of crossing of the mesenteric root. Fixation of the cecum and ascending colon is usually defective and therefore leaves a mobile cecum which is liable to torsion with resultant obstruction and possible infarction.

Lastly, the second stage of rotation may lead to a variety of defects due to congenital bands that may obstruct either small or large bowel.

Third Stage. The third stage of rotation is completed about the time of birth. This stage concludes the rotation of the cecum and ascending colon from the subhepatic region to their normal positions in the right iliac fossa and right gutter respectively. Fixation of cecum, right colon and third and fourth portions of the duodenum also takes place during this final stage (Fig. 30).

PATHOLOGIC CONDITIONS. If the cecum fails to descend from the subhepatic region, three things may result: first, appendicitis in the right upper quadrant; second, the cecum in an attempt to fix itself to the lateral wall forms congenital bands across the duodenum which in turn may obstruct the duodenum; third, the cecum may become fixed laterally across the duodenum, but the small intestine mesentery may not become fixed, which may result in a volvulus of the small intestine about the superior mesenteric artery as an axis.

If the cecum descends normally, two pathologic conditions may still result: (1) a retrocecal appendicitis; (2) a nonfixation of the cecum and ascending colon with resultant volvulus. In the last condition the symptoms may be those of obstruction or those of vascular accident with infarction of bowel. The pathology resulting from defects of third-stage rotation may become apparent at any age.

Treatment

It is not within the scope of this communication to discuss surgical techniques. Many of the obstructions of the bowel due to defects in rotation are easily corrected by cutting congenital bands and unwinding twisted loops of bowel. When bowel is infarcted, resection is necessary, and in the infant this adds greatly to the morbidity and mortality. There is one rule the surgeon must abide by after the unraveling of the twisted misplaced loops of malrotated bowel, and that is, he should never try to rotate the bowel into normal position or try to fix it to the parietal walls.

In conclusion, it is suggested that it behooves the pediatrician and the surgeon to understand the process of bowel rotation in order that they may recognize the pathologic state when confronted with it and, furthermore, know how to unravel it at operation.

DUPLICATIONS OF THE BOWEL

Gross has made a plea for standardization of the term "duplication" of the intestinal tract. Pediatricians and surgeons interested in this condition should heartily agree. Many terms have been used to designate a structure that is attached to some portion of the alimentary tract, is cystic, contains serosa, muscularis and mucosa, and may be spherical or may duplicate a portion of the alimentary tract. Some of these terms are "enteric cysts," "inclusion cysts," "enterogenous cysts," "ileum duplex," "reduplication" and "thoracic cysts."

Duplications may occur anywhere along the alimentary tract. They must duplicate some portion of this tract, but not necessarily the por-

tion to which they are attached. Duplications may have a common wall with the normal tract, they may be tubular appendages attached at one end to the alimentary tract, or they may have an independent mesentery. The first type is most common. Duplications are found most commonly along the ileum and ileocecal region; the next most common region is within the thoracic cage; the third most common region is the colon and rectum; the rest are found along the jejunum, duodenum and stomach. Many of the thoracic duplications are derived from the stomach, duodenum and ileum. Colon duplications have been found in the chest.

Embryology

There are many theories to explain the origin of duplications of the alimentary tract. None has as yet satisfactorily clarified the issue. Space does not allow discussion of the many theories.

Clinical Picture

It is difficult to state what the incidence of occurrence is, since the age when symptoms develop may be from infancy to old age. Further, many patients may die never having had symptoms severe enough for operation. Therefore it may be stated that duplications are probably not rare. Males and females are about equally affected.

There are three common symptoms. The most common one is obstruction due to expansion of the cystic duplication with resultant pressure on the adjacent tract. Pain is the second most common symptom. This is caused in the main by distention of the cyst wall. Hemorrhage is the third in frequency and may be the presenting symptom. Hemorrhage results from erosion due to pressure or frank ulceration of the mucosa. There also may be hemorrhage into the cyst proper with sudden pain and occurrence of a palpable mass.

Since the colon is the third most common site for duplications, obstruction due to a duplication need not be exceptionally rare. Certainly when an obstruction of large bowel is found in childhood, one must consider the possibility of a colonic duplication, particularly if the barium enema appears to represent extrinsic pressure. Furthermore, duplication of the cecum or ileocecal region may give rise to intussusception. The typical picture of intussusception is seen in these cases, the diagnosis of duplication being made at operation. The colon is also the most common site for complete duplication or double-barreled colon. This latter type usually does not cause obstruction, since both tubes carry feces. However, it usually has a complicating fistula to either the genitourinary tract or the perineum.

Treatment

Treatment, once a complication arises or once a diagnosis is made, is obviously surgical. Resection of the involved region is indicated, with restoration of continuity of the bowel. Double-barreled colon requires resection with reconstruction, which at times may be extremely difficult but necessary.

CONGENITAL MEGACOLON

Primary or congenital megacolon may be defined as a great dilatation, elongation and hypertrophy of a portion of the colon or at times the entire colon, associated with retention of enormous amounts of feces and an absent defecation reflex. It is due to a diminution or more likely a complete lack of parasympathetic ganglia, usually in some segment of the distal colon.

Etiology

Hirschsprung[14] was not the first to describe this condition, but in 1886 he did give us the most classical description. Hirschsprung described the clinical study and autopsy findings of two patients with megacolon. There is little to change in his description of the disease except the cause. Hirschsprung thought the defect was in the dilated hypertrophied portion of the colon and not in the narrowed segment.

Dalla Vallé[3] was the first to suggest that congenital megacolon was due to an absence of the ganglion cells of the myenteric plexus. Dalla Vallé's interpretation was first published in 1920, and in it he described the microscopic findings from sections of dilated hypertrophied colon and the narrowed sigmoid colon. The narrowed segment of colon in authenticated cases had no ganglion cells.

The site of the aganglionosis is most commonly in the region of the lower sigmoid and rectosigmoid. However, the entire sigmoid may be involved, as may be the lower sigmoid and entire rectum. There also may be skip areas in the sigmoid and rectum. Aganglionic segments have been described not only in other portions of the colon, but also in the pylorus of the stomach and in the cardio-esophageal junction. (Figures 31 through 34 demonstrate the findings in two of the author's cases.)

Pathologic Physiology

The pathologic physiology of megacolon is as follows: There is always a reduction or complete absence of ganglion cells in the myenteric plexuses of the involved area. There is no diminution of the nerve

Fig. 31.

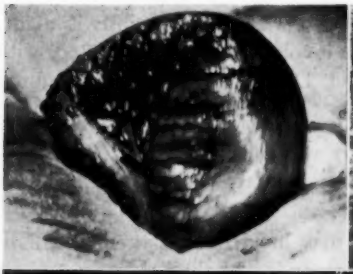

Fig. 32.

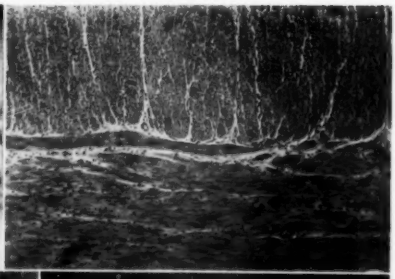

Fig. 33.

Fig. 34.

Fig. 31. The sigmoid colon of a 9 months old girl who had considerable difficulty with bowel movements from birth. The figure shows the tremendously dilated upper portion of the sigmoid colon and the narrow lower portion of rectosigmoid. Picture was taken in the operating room.

Fig. 32. Microscopic section of the narrowed segment pictured in Figure 31. There are no ganglia between the longitudinal and circular muscle layers.

Fig. 33. Microscopic section through the dilated hypertrophied colon above the narrowed segment pictured in Figure 31. This is the normal distribution of ganglia between circular and longitudinal muscle layers of the colon.

Fig. 34. The rectosigmoid unit of an 8 weeks old girl. The narrowed segment was easily felt with the examining finger at 3 weeks of age.

fibers. According to Cannon,[2] peristalsis cannot take place in the bowel in which the myenteric plexuses have been destroyed. The myenteric plexuses contain the ganglia of the parasympathetic division of the autonomic nervous system. Most of the colon receives its parasympathetic supply from the vagus nerves. Pick[17] has shown that vagus branches pass well beyond the midtransverse colon on down the descending colon. The distal colon receives its parasympathetic supply from the pelvic division. When the vagus and the pelvic parasympathetic supply are intact, progressive peristaltic waves move throughout the colon to the anal canal. Failure of the parasympathetic ganglia to develop explains the failure of progressive peristalsis across the involved segment of distal colon. The dilated colon in megacolon contains normal parasympathetic myenteric plexuses. Therefore treatment should

be directed toward the distal colon, including the rectum, and not toward the dilated hypertrophied colon above.

Clinical Features

Incidence. Megacolon is said to occur once in 9000 births. Males are affected four or five times more commonly than females.

Symptoms. There are two cardinal features: the difficulty at stool from birth and the progressive enlargement of the abdomen. If the

Fig. 35. Barium colon enema done on the patient represented in Figures 31, 32 and 33, showing clearly the same narrowed segment as found at operation.

condition is allowed to progress and laxity in bowel care continues, anorexia, lassitude, and finally malnourishment result. On examination the abdomen is protuberant and tense, and if disease is long-standing, the skin is thin, tense and shiny. Rectal examination usually reveals an empty ampulla. In the infant the spastic segment many times can be felt with the examining finger. Sigmoidoscopy demonstrates the narrowing if it is within the length of the sigmoidoscope.

Treatment

There have been many types of treatment advised for megacolon, including special diets, laxatives, enemas, drugs such as Mecholyl and

Syntropan, spinal anesthesia, lumbar sympathectomy, and resection of various portions of the colon. None has proved uniformly successful.

Swenson[19] in 1948 first described a successful operation for megacolon. He reported satisfactory results one year later on 23 cases.[20] Since Swenson's original description there have been several modifications, e.g., Hiatt[12, 13] and State.[18] The Swenson technique is considered far superior to the others by most surgeons performing surgery for megacolon. This technique combines an abdominal and a perineal approach. The major portion of the narrowed segment plus several centimeters of dilated sigmoid are removed through the abdominal approach. The distal rectum is then intussuscepted down through the anal canal, after which the closed upper sigmoid colon is introduced through the inside-out rectum. The excess rectum is excised and the anastomosis is done outside the anal canal. For a complete description of this procedure the reader is referred to Swenson's several articles on the subject.

Summary

In summary, the history of congenital megacolon has been briefly reviewed, the pathologic physiology discussed, the clinical picture presented, and the recommended therapy suggested.

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130 East 79th Street
New York, New York

NEOPLASMS OF THE COLON AND RECTUM IN INFANTS AND CHILDREN

A Survey of Polyps, Polyposis and the Uncommon Malignant Lesions of the Large Bowel

W. H. SNYDER, JR., M.D.

LAWRENCE CHAFFIN, M.D.

M. H. SNYDER, M.D.

Neoplasms of the large bowel in children are of infrequent occurrence, but the literature shows increasing numbers reported in late years. By far the commonest of these lesions is the so-called polyp or adenoma, and this discussion is concerned chiefly with the problems arising from it. It would be of value to be able to quote statistics as to the rate of occurrence of polyps, but such figures as there are vary so widely with different authors that only an approximation can be given. Pennington[62] quotes Bokay, who in 1868 reported that among 58,970 children he found only 25 polyps of the rectum or large bowel. Jacobi[42] stated that he had seen 100 cases in children two to five years old, in the course of his surgical career. Kennedy[44] reports on 49 seen at the Mayo Clinic in 15 years. Helwig[39] estimates the rate in children at about 3 per cent, and Turell[76] says it occurs in about 2 per cent of persons under 45. However, Kerr[46] found polyps in 100 out of 349 children with rectal complaints, and Atwater and Bargen,[3] examining entire colons, mainly from adults, at autopsy with a magnifying glass, placed the incidence at 69 per cent.

From the Department of Surgery, University of Southern California School of Medicine, and the Los Angeles Children's Hospital, Los Angeles.

ANATOMY

A brief discussion of the anatomy of this region (Daniels[21]) in infants and children is pertinent, since there are distinct differences from that of the adult. In the newborn and infant up to three months of age the structures are distensible and very elastic. The plane of the pelvis is nearly vertical, and the rectum is but loosely attached to the sacrum. The sacral promontory is less marked and lies relatively higher up. The rectum is only about 2 to 3 inches long in the newborn, and its lumen about one fourth that of the adult rectum. Rapid growth takes place in the next two years, making the lumen one third to one fourth greater than at birth. The next 10 years are a period of slower growth, and it is not until the end of the second decade that the lumen has reached its adult proportions. The lateral ligaments are poorly developed and low, compared to those of the adult. The anterior reflection of the peritoneum is low, at about the level of the fourth sacral vertebra. Since this results in a deep rectovesical and rectovaginal pouch, these relations should be borne in mind in operative procedures in infants. The flexibility and distensibility of the anal canal in infants allow for a bulky stool and by the same token will allow passage of a well lubricated index finger in children two or over and the little finger in those younger. One should bear in mind also that the sigmoid is very tortuous in the infant. At birth it is pushed up out of the pelvis altogether by the rectum filled with meconium. It is complex and redundant and may have an axis to the right or left in the pelvis and may rise high into the abdominal cavity. This makes the examination of the sigmoid and colon in infants and young children difficult and, at times, dangerous.

POLYPS

The term "polyp" is loosely used, but suggests to the surgeon a certain gross structure which is rounded, protruding and either sessile or pedunculated. Microscopically, a polyp may represent any one of a variety of histologic structures, but most are found to be adenomas or papillomas. In children, adenomas are the type usually found; papillomas rarely. Bacon[4] shows one papilloma in his textbook in a two year old child. Papillomas, as described by Sunderland and Binkley,[73] are tumors of the surface epithelium of the mucosa with secondary glandular changes. They are soft and spongy and difficult to feel. Adenomas, on the other hand, arise from the mucosal glands with little if any involvement of the surface epithelium. They vary in size from a few millimeters to several centimeters. Those found in children have a somewhat different microscopic appearance from those of the adult. The childhood type contains a larger amount of connective tissue in

which are numerous eosinophils (Castro[14]). Of adenoma of the rectum, Boyd[11] says that its most interesting characteristic is its large incidence, for most cases occur in children. Ackerman[1] calls the childhood type "cystic or retention" polyps and says they "do not become malignant." All show some degree of inflammation, edema and hemorrhage. The consensus, however, is that in any age group these tumors are essentially alike clinically and should be treated identically (Helwig[39]). Although the great majority of these polyps are true adenomas, other types of neoplasms do occur in polypoid form. Dewis[24] in an account of 101 rectal lesions found 81 to be adenomas; the remaining were divided as follows: two angiomas, two fibromas, 10 myomas, six lipomas, disregarding the ages.

Age Incidence

Gross[35] believes that adenomas occur most frequently in the first 10 years of life. The number seems to fall off during the second decade for unexplained reasons, but from that time on they increase in frequency throughout life. Radke[65] reported one case in an infant four months old, and Smith[71] reported one in a girl baby of six months. Kennedy's youngest was six months, and Kerr's nine months. This would suggest a condition existing at birth or rapid growth after inception. Hall[37] found one in a child one year old, and others have reported them in children of 18 months. These are the youngest we have found reported, but, as previously stated, the greatest number in children occurs between two and five years. In Kerr's series 98 out of 100 were in children under 11 years of age. Kennedy's series of 49 showed 28 to be five or under and 21 six or over, the oldest being 13, although in a later series[45] he reported one of 14. Though the incidence is lower in the second decade, polyps do occur in that age group, as in Kennedy's series. Sandusky and Parsons[67] found 18.6 per cent in the first decade, 7.1 per cent in the second decade, and 5.1 per cent in the third decade. The average age among the children's series is about three years (Kerr[46]). In those found at older ages there is frequently a history of symptoms lasting over a period of years, indicating their onset at an early age. Harris[38] wonders why their scarcity in the second decade. What becomes of them? This may be explained in part by self-amputation.

Etiologic Factors

The etiology of the condition is obscure. There is no doubt that in later life some arise on a basis of chronic inflammation, but this does not seem to apply to those of childhood, nor to most single or scat-

tered polyps found at any age. Kennedy believes the condition to be congenital. Swinton and Warren[74] think that these are true tumors due to an inherent defect in cell growth. Kerr advances the theory that they are mucosal rests (Cohnheim theory) trapped in scar or inflammatory tissue and stimulated to growth by the same forces which activate the healing process. Being raised above the level of the mucous membrane, they then become subject to trauma. Trauma is undoubtedly a factor in the course of the existence of the polyp. Most are pedunculated in children, and the larger they are, the longer the pedicle is likely to be. Some are several inches in length. The sequence of events seems to be that, as the tumor enlarges and protrudes into the lumen of the bowel, peristaltic activity pulls on it and elongates the pedicle.

Mucosal redundancy is common in these children with polyps, and it is possible that chronic constipation and faulty bowel habits predispose to polyps. Church and Schwartz[17] found that 10 out of 30 children with prolapse had polyps.

The location of the polyps is of interest. The majority are found in the rectum (75 per cent according to most authors), or so low in the sigmoid that they can be seen easily through the sigmoidoscope. Many can be found on digital examination alone. Swinton and Doane[75] found 184 out of 331 in the rectum or anus. Buie[12] found 56 per cent in the rectum, and Castro, 77 per cent. Gross says that in children three fourths are in the rectum. Because more than one polyp is found in at least one third of the patients, the finding of one makes search for others obligatory. In Kerr's series of 100 children 68 had a single polyp, and 32 had more than one. In Gross' series two thirds had a single polyp, and one third, two to nine; 10 had multiple polyposis. Turell[77] says that 10 per cent of those with adenoma in the rectum also have adenoma in the colon.

Symptoms

The symptoms vary from none at all, the condition being discovered incidentally or at autopsy, to severe abdominal distress. Rarely, the first thing which brings the condition to the attention of the physician is acute *obstruction* due to intussusception, caused by the polyp. Occasional reports are made of intussusceptions caused by a polyp.[34, 79] However, the commonest, indeed almost constant, symptom is that of painless *bleeding* from the rectum. The stool may be streaked or covered with blood, or there may be blood mixed in with the feces. If it is high in the colon, parents may not know of it, as it may well be occult. Bleeding varies greatly in amount and frequency. There is usually only a teaspoonful or perhaps the passage of small clots, but in rare cases rather severe hemorrhage occurred. In Kennedy's series one out of 11

needed transfusion. This was probably due to the sloughing of a polyp from its pedicle, which then bled profusely. Bleeding may occur every day in small amounts, or it may appear at intervals of several weeks or months. This bleeding is probably the result of irritation, even ulceration of the polyp by the passage of hard stools.

Depending upon the degree of interference with normal functions, there may be some *abdominal distress* and *diarrhea*. In Kerr's series 19 per cent complained of diarrhea. Not uncommonly the mother first sees a mass protruding from the anus. In some instances a mass is passed. The mother may recover it and bring this to the attention of the doctor. Self-amputation occurs frequently in children, but rarely in adults. Kerr cites 19 cases of self-amputation in his series of 100 polyps in children. Out of Harris' 70 cases eight amputated themselves. Protrusion of the polyp from the anal orifice is not rare when the polyp is situated near the outlet, but it also occurs when the polyp is situated 10 to 12 cm. above the outlet. In Kerr's series protrusion with intussusception occurred, and it was found that the polyp was located 25 cm. above the outlet. Stockman[72] says that prolapse of rectum accompanying polyp occurs more often in his experience than mentioned by other authors. Polyp may be considered a cause of prolapse in one child out of three. Ten out of 33 cases of prolapse had polyps in Neuhoff's series.[60]

Abdominal distress varies with the size and location of the polyp. Kerr states that in every child who had a polyp situated 25 cm. or higher in the intestine, this was a constant complaint in differing degrees. When found high in the colon, according to Baffes and Potts,[6] pallor, weakness and anemia may be presenting symptoms.

Diagnosis

The diagnosis may be suspected from the history of painless bleeding and verified many times by the appearance of a mass at the anal orifice. Ordinary rectal examination will reveal most polyps. Examination in a squatting position will allow for palpation higher up. The finding of a single polyp demands further search for additional polyps, since one third of patients have more than one. Examination with a proctoscope is perhaps best performed under a general anesthetic, as advised for the treatment of this condition by Gross, Daniel, and Arnheim,[2] but others find that the examination of even small children is done safely and satisfactorily under heavy sedation.[33, 59]

Infant proctoscopes are usually about ½ inch in diameter and 10 inches in length. They are not very satisfactory instruments to work through and are unnecessary after the age of two years (Harris[38]). In children beyond this age the adult proctoscope is a much more usable instrument.

A means of adequate lighting, a bulb and connection for inflating are a part of most modern proctoscopes. A suction tube is an essential, not only for removing fluid, but also to eliminate fumes which may cause explosions (Carter[13]). Those made of nonconducting bakelite or with a sheath of this substance (Montague) seem preferable to metal ones when using electric current for coagulation. This eliminates the necessity for encasing the snare and forceps in rubber tubing when a coagulation current is applied.

Turell says that the inverted jackknife position is best. However, many feel that a proctoscopy should be performed with the patient in a lithotomy position, since most adenomas are on the posterior wall. A knee-chest position may so distort the relationships in a child that the polyp cannot be seen. Barium enema was found not a reliable diagnostic medium when the lesion lay in the distal 25 cm. of the large bowel (DeMuth et al.[23]). Repeated examination may be necessary.

If symptoms are marked and debilitating, further search must be carried on until the presence or absence of polyps is established. This may be difficult. Careful preparation of the bowel is most important. The barium and air, double contrast technique is usually recommended for accurate visualization of polyps beyond the reach of instruments. However, the bowel must be thoroughly cleansed of gas and fecal material which would obscure the appearance of small polyps. Kennedy recommends castor oil as the one satisfactory drug for this purpose, giving 15 cc. to infants and 30 cc. for older children. It is given on the evening before the examination, and dinner is withheld. The next morning at least an hour before the examination is to be done, a small enema of about 250 cc. of saline solution is given to remove the last remaining bits of material. Breakfast may then be given.

As the opaque enema is given, it is carefully watched as it ascends into the colon. With palpation and rolling the patient from side to side, the whole colon can be visualized. This must be done quickly, for the patient will be most uncomfortable and probably uncooperative. When the enema has been evacuated, a further examination may reveal small irregularities on the surface of the mucous membrane of the bowel which may be identified as polyps. According to Gianturco,[32] an increase of voltage will bring out into sharp relief the existing polyps in the wall of the colon. Also, Robinson[66] says that a barium enema made translucent by high kilowattage and the use of spot films is the best method. The technique of double contrast media is very tricky.

Differential Diagnosis

A few words should be said about differential diagnosis in the case of bleeding by rectum in infants and children. Local lesions may be

rather readily eliminated as a cause of the bleeding. Fissure, ulcer or abrasion is apparent on close scrutiny of the anal region. Also these lesions are accompanied by pain and discomfort on defecation, which is not the case in polyps. Prolapse can be identified by the appearance of the pale pink mucosa, a polyp being of a port wine color. Prolapse, of course, may accompany the presence of a polyp, as in Neuhoff's series. Such causes of bleeding as ulcerative colitis or dysentery are accompanied by debility and wasting, whereas the child harboring a polyp is usually well nourished. The bleeding accompanying Meckel's diverticulum, coming as it does from higher in the intestinal tract, is older, darker blood and well mixed in with the stool. Intussusception makes itself recognized by the severity of the symptoms accompanying the bleeding.

Many polyps exist without causing any symptoms whatsoever. In Helwig's series five patients were found in the course of 449 autopsied under the age of 20. None had had symptoms suggestive of polyp. Blood dyscrasias causing rectal bleeding are often accompanied by obvious systemic manifestations or can be recognized by laboratory studies—blood picture, prothrombin time, bleeding and clotting times, bone marrow studies, and so on. Scurvy as a rare cause of bleeding must be mentioned.

Malignant Potential

The question of the malignant potential of these childhood polyps arises naturally, since the adult type of polyp is generally conceded to be a precursor of malignancy. Kennedy, according to the Broder tissue classification used at the Mayo Clinic, considered about 55 per cent of his series to be adenocarcinoma. However, other authors such as Helwig failed to find any evidence of malignant degeneration. Two of Kerr's patients who had a long history of polyp were proved to have adenocarcinoma of the bowel at early ages (one at 12 and one at 24). Oosting[61] also reported adenocarcinoma found in a rectal polyp in a boy of eight; Bacon,[5] in a boy of six. Marion and Avet[54] report a boy of four with two polypoid growths diagnosed as cylindric epitheliomas, and Schilla[70] reported carcinoma found in a polyp in a two year old child, confirmed by many pathologists. Gross found carcinoma in a polyp in a two year old. In none of these reported cases, however, is there clear evidence of an invasive carcinoma, and yet it is reasonable to surmise that it would have developed had the polyps not been removed. There is thus sufficient room for suspicion of potential malignancy in childhood polyps to make careful sections of the extirpated tissue advisable.

Treatment

Treatment consists in removal when found and correction of whatever existing conditions may seem to predispose to polyp formation: constipation, faulty bowel habits, prolapse and redundancy.

The rectum and lower sigmoid are easily visualized and inflated with air, the polyp and its pedicle identified. Treatment of a single polyp is simple if the tumor can be prolapsed through the rectum. The pedicle is ligated and the tumor removed, or a snare (Daniels,[21] Gross,[35] Castro[14]) is placed at the base of the pedicle. This is coagulated and the polyp removed for microscopic study. If somewhat higher, but visible through the proctoscope, it can be grasped with a tenaculum and turned so that the pedicle can be seen. Direct fulguration (Kennedy, Harris) of some pedunculated and all sessile polyps is accomplished by grasping them with the alligator forceps and applying the current directly, or by the use of the double loop resector (Turell[78]). The ball-tipped electrode is commonly used for fulguration. In all these procedures great care should be taken not to damage the bowel wall. Boehme[9] uses a suction cautery which simultaneously removes the smoke from the field and holds the polyp away from the bowel mucosa. This aids in preventing inadvertent cauterization of normal mucosa and possible bowel wall perforation; however, it does not preserve a specimen for microscopic examination. Turell also advocates extirpation by means of the cold biopsy forceps followed by thorough desiccation of the base. Harris feels that the use of the snare should be confined to polyps below the peritoneal reflection. Above this level he uses direct fulguration and feels that in most cases enough of the polyp can be left intact to obtain a satisfactory biopsy. Lesions below the peritoneal reflection, Turell feels, are safely treated in the office. In those higher up the risk is greater and the patient should be hospitalized. If a polyp has sloughed off, the area from which it has come should be examined first for continued bleeding, secondly for incomplete separation of adenomatous tissue which may provide a nucleus for regeneration of polypoid tissue. Of course these patients should be watched for occurrence of further polyps.

Tumors higher in the intestine must be approached via colotomy, incision being made lengthwise in the antimesenteric border. For short pedicled and sessile polyps, wide resection of the mucosal base and submucosal structures is advocated. For "carcinoma in situ," or with no invasion of the pedicle seen, simple excision is usually safe. However, Scarborough[69] reported three in adults showing no invasion of the pedicle, but in which metastases were demonstrated in regional glands. Some adult clinics advise immediate microscopic study of the specimen to determine whether invasion has taken place. In such a case or in the case of large sessile polyps, resection is done with end-to-end an-

astomosis, with or without colostomy or cecostomy. Welch[82] advises that simple polypectomy for polyps of the colon be abandoned in favor of segmental resection of the colon. He gives figures from the Massachusetts General Hospital of 70 polyps in adults which looked benign through the sigmoidoscope or colotomy incision; 11 were found to contain cancer. He argues that removal of polyps from the rectum through an instrument is justifiable since the area can be kept under surveillance. Higher up, they can be observed by a second laparotomy only. He feels that since the operations of polypectomy and resection are of equal magnitude, the second is far preferable from the long-range viewpoint. His series does not include children, of course, and although the likelihood of malignant degeneration is less, this cannot be entirely ignored.

Thus far we have dealt with single or occasional polyps. As we have brought out, more than one occurred in about one third of the cases in most series. A term useful for the designation of this latter picture is that of "scattered polyps." They may occur at any point in the colon and must be treated according to the location at which each is found.

Summary

From certain theoretical standpoints, polyps of the colon constitute a different disease in children from that in adults. Among reported series of juveniles their frequency reaches a peak at three and one-half years. They tend to be self-limited by means of extrusion and auto-amputation. Invasive malignancy in a microscopic section is unknown, but cellular changes indicative of malignancy occur on rare occasions, and almost conclusive circumstantial evidence of progression of a polyp to metastatic carcinoma has been reported on two occasions in the world's literature.

POLYPOSIS OF THE COLON

Diffuse polyposis or adenomatosis was first reported by Menzel in 1721. It was described by Virchow in 1863, but the first recognition of its familial predisposition was recorded by Cripps[19] in 1882. It is fortunately a rather rare condition, but as attention has been focused on it, increasing numbers are reported.

There is considerable confusion in classification. Erdmann and Morris[28] described two types which they called "adult" type and "adolescent" type. The former is generally considered a secondary type following inflammatory disease. But the type we are concerned with here—or the Erdmann–Morris "adolescent" type—is of unknown origin. It has been proved conclusively to be hereditary or familial. Some

authors, notably McKenny,[52] consider it to be truly congenital, since they have found instances of it early in life. Most authors, however, find that it manifests itself most commonly in the second decade and is found with increasing frequency thereafter. Examples of many instances of it in a single family are growing in numbers.

Lockhart-Mummery and Dukes[49] have contributed largely to studies of the familial aspects of this disease. Dukes[26] in 1930 published 13 family pedigrees demonstrating the hereditary nature of the condition. It seems, at times, to act as a genetic dominant, at other times as a recessive characteristic. Probably it represents a gene mutation which, if carried as a mendelian dominant, appears in each succeeding generation; if as a recessive, it may skip a generation unless both parents carry the gene. This makes it difficult to trace in the course of the few generations available for study. The nature of this gene is speculative; it appears to be a tendency of the subepithelial tissue, and secondarily the epithelial cells of the mucosa itself to proliferate at a certain age with resultant formation of innumerable polyps throughout the large intestine. The inheritance of this disease shows no sex linkage; it has been transmitted from mother to son, father to daughter, and so on. The distribution between the sexes is fairly even. In those families in which it occurs there are a significant number of cases of cancer of the colon or rectum, with or without associated known polyposis. When carcinoma does occur, it is at an earlier age than would otherwise be expected.

Relation to Peutz-Jegher Syndrome

Mention should be made of a related syndrome, occurring even more rarely but definitely hereditary, and that is the so-called Peutz-Jegher syndrome. In this disease, as first described by Peutz,[63] polyposis of the small intestine was found to be associated with a peculiar oral melanosis in certain patients and to occur in family groups. This melanosis consisted of bluish to dark brown spots found on the lower lip and buccal surface of the mouth. In reporting such cases as he could find in the literature in 1949, Jeghers[43] included six cases in which polyposis was present in the colon. There seems some likelihood that this is a variation of the same disease. Wolff[84] states that polyposis is always present in the small intestine and may also be found in the colon. In a case reported by Crumpacker[20] the colonic disease seemed to require treatment most urgently, and at the last report he had never determined whether or not there existed polyps in the small intestine.

Histologically, the appearance of some of these involved bowels differed from each other, but probably in the matter of degree. One type frequently encountered shows hundreds of polyps of great size

variation, some sessile, some pedunculated, arising from normal-appearing mucosa. Another type—probably an extreme degree of the same disease—shows polyps superimposed on hypertrophied mucosa so that no normal mucosa can be identified between the polypoid growths. This type of polypoid growth will recur even if removed by fulguration. Most authors feel that the relation of this disease to eventual cancer is undisputed. The malignant potentialities of each polyp multiplied by the enormous number of polyps present in these cases make the chances of carcinoma heavily weighted against the patient. Mayo and co-authors say without hesitation that every patient with untreated polyposis of the colon will die of cancer or have advanced malignancy by the age of 50. It is rather common to find malignant degeneration in one or more of these polyps when first seen. Various authors report percentages of carcinoma already present in patients with polyposis as from 26 to over 40 per cent (Hullsiek,[41] Dixon and Benson[25]). Malignant degeneration is found even in children. Neel et al.[56] say that because of the wide variation in age of development of this condition in young people and children, the age of 25 should be considered within the danger zone; 12.5 per cent had cancer before that age.

Age

As to the age of appearance of this disease, most authors find it becoming evident at puberty. However, there are numerous reports of its occurrence at a much earlier age. Mayo's[55] series ranged in age from three to 56 years. McKenny followed a family in which all four children were afflicted. The youngest was two years old; the next ages were five, nine and 11. The size of the polyps found was slightly larger at each older age level, seeming to demonstrate the appearance either at birth or at a specific early age, and steady progression as the child grew older. As far as this author can determine, it has not yet been found at birth, to prove it to be congenital, but such a series as this of McKenny's is suggestive. Dixon[25] saw two patients each 18 months of age. LeFevre and Jacques[48] reported it in a four month old child, the youngest we have found in the literature.

Symptoms

Most commonly the symptoms are diarrhea, passage of mucus and blood, pain in the lower abdomen and, frequently, tenesmus. The history of these complaints may go back over a period of years. Acute obstruction may be the presenting symptom; intussusception has occurred in some of these patients caused by one of the large polyps (Bokay,[8] LeFevre). Some patients are symptomless and are found only during investigation of members of a polyp family.

Diagnosis

The diagnosis is established by digital examination and sigmoidoscopy. The condition will be recognized by the multiplicity of the lesions. Eosinophilia is present in some cases. The extent of the involvement can be determined only by roentgenographic studies with double contrast techniques. It is interesting and important to note that though the pathologic involvement may seem limited to certain segments of the bowel, it may later extend to include much more or all of the colon and rectum (Black et al.,[7] Hoxworth and Slaughter[40]).

Management of Familial Polyposis

These patients are too few and have been observed over too short a time for a definitive program of treatment for the condition to have evolved. Eventually all disease-bearing areas of the colon must be removed. The disease is progressive and will terminate fatally because of inanition or malignant degeneration without treatment. McKenny[51] reported favorable results with palliative x-ray treatment in a few children. But there was no follow-up report by which long-term results might be judged. It offered relief temporarily to patients who were poor surgical risks.

Surgery offers the only hope of cure. The problem is how early to operate and how extensively to resect. Each case must be individualized. Baffes and Potts report a six year old boy with such severe debilitating diarrhea that total colectomy was indicated. Preliminary fulguration of rectal polyps was accomplished at several sittings; then total colectomy with ileoproctostomy was carried out. He was in good health 11 months later. Children with symptom-free polyposis may probably be safely observed. Clark and Parker[18] discuss a family of three adolescent children with symptom-free polyposis. The grandmother had died at 41 of cancer of the colon, and the mother at 34 of the same disease. The decision of Clark and Parker was to watch these children until they reached their middle twenties. However, with reports of death at 15 (McKenny) and at 19 (McCarty[50]) from carcinoma arising on a basis of polyposis, there is a certain risk in waiting.

It is Cattell's[15] belief that total colectomy with abdominoperineal resection is the only satisfactory method of treatment. He feels that since the rectum carries the greatest number of polyps it is unwise to leave it. However, Mayo et al. feel that in a young person it is important to give him as nearly physiologic function as possible. Therefore he leaves the lower portion of the rectum whenever possible, fulgurating the polyps, of course, and keeping the patient under close surveillance for the rest of his life. Examination should be done at least every six months and preferably every three months. Fulguration

must be repeated whenever new polyps arise. This may lead to a further complication of scarring with stenosis necessitating dilatation. When no normal mucosa can be seen or, of course, when cancer is present in the rectum, there is no choice but abdominoperineal resection.

In leaving the rectum, it is important that no more be left than can be easily visualized through the proctoscope. It has not been established whether or not carcinoma can develop when all polyps are fulgurated as they appear. Certainly cancer has appeared more than once in the rectal stump (McCarty and Guptill[36]). In order to preserve the sphincter, yet eradicate as much of the rectum as possible, Ravitch[67] has done anal ileostomies. This procedure is not always satisfactory, however, and a perineal stoma is more difficult to care for than an abdominal stoma. When colectomy is elected, it must be determined whether it can be done in one stage or will require more. From the viewpoint of the patient and his family a one-stage procedure is preferable, when possible. In recent years, with modern advances in anesthesia and the use of antibiotics and chemotherapy, this has been done successfully a number of times.

When instances of polyposis come to the attention of any physician, he should make every effort to study the remainder of the patient's family both for the purpose of treatment at the optimum time and to assist in genetic studies of this disease. As many authors have found, it requires tact and persistence. Every plan of treatment should be considered from the viewpoint of the psychologic effect of this treatment on future patients in the same family.

Summary

Polyposis is a hereditary disease appearing usually at or about puberty, though we have noted certain exceptions here. It progresses in the absence of treatment to carcinoma in most cases unless death from other causes supervenes. The only cure is by total colectomy. If anything less is done, the patient must be kept under constant supervision for recurrence and malignant degeneration. Ileoproctostomy with close observation and removal of polyps occurring in the anal stump seems indicated for polyposis in children. McKenny suggested sterilization of members of polyposis families, but nature takes care of this problem, because most families die out of their disease.

MALIGNANCY OF THE LARGE INTESTINE IN CHILDHOOD

Cancer in childhood has become an important public health problem and now ranks third among causes of death in children between

the ages of 10 and 14.[80] However, carcinoma or other malignancy of the digestive tract is one of the more rare. Potts[64] reports that at Children's Memorial Hospital in Chicago no child under the age of 13 years has ever been seen with cancer of the colon. However, Fowler[30] reports 21 cases of cancer of the large bowel in young people under 25. Williams[83] collected 50 cases of carcinoma of the colon in children under 16 from the literature and added three of his own with a fourth mentioned. He states that it can occur at any age, but the youngest he found was three. Sporadic cases (Chaubenet[16]) keep appearing in the literature until, on review, a sizeable number can be found. Bonelli[10] cited 142 cases of carcinoma of the gastrointestinal tract in children from the literature, of which 73.8 per cent were in the descending colon and rectum. Nelson's textbook[58] reports from the literature 30 authenticated cases of sarcoma or carcinoma of the rectum and colon in children under 12. Nelson et al.[57] report a case of their own, in a boy of 14, and cite six more from the literature in children under 17.

Types of Neoplasm Found

A number of reports have been made of carcinoma arising in an otherwise benign polyp, and these we have dealt with in the section on Polyps.

Epithelial tumors are most common, but sarcomatous growths are occasionally reported. Bacon reports a boy of three with a reticulum cell sarcoma of the rectal wall. Galloway and Owens[31] reported a case of lymphosarcoma of the appendix in a boy of six who finally succumbed to general sarcomatosis, and added seven more such cases from the literature. Erbes and Stehlin[27] report lymphosarcoma of the cecum with intussusception in a boy of nine. Bonelli found reports of 13 cases of sarcoma of various types in the large bowel and rectum. Other reports found by this author dealt with various types of carcinoma. Ewing[29] mentions two reports of a gelatinous adenocarcinoma in the ascending colon of a boy of 17, and the splenic flexure of a boy of 13.

Diagnosis

The diagnosis is seldom made preoperatively—it is not thought of in obscure complaints related to the bowel. The diagnosis of acute appendicitis or tuberculous peritonitis is usually made. If a disorder satisfies all but a few criteria of some common illness, an effort should be made to eliminate malignancy. Most authors stress that in operating for appendicitis and finding inadequate lesions to explain the symptoms, search for other abdominal disease should be made and

malignancy not ruled out without careful search. Dargeon[22] and others who write of malignancy in children emphasize that vague and persistent complaints related to the abdomen should put the physician on guard against malignancy, particularly in children who show an unusual growth pattern. Those of generally retarded growth or those with precocious development are most prone to some type of malignancy.

Symptoms

The symptoms of malignancy of the bowel in children do not necessarily follow the adult pattern. One of the commonest is pain, which is not seen until late in adult malignancy of the bowel. The pain is usually described as intermittent and colicky, occurring after meals and relieved by passage of gas or stool. There is a difference in symptoms, depending upon what part of the bowel is involved. In the right half, as in adults, there may be anemia as a result of ulceration and bleeding. Because the right colon is fixed, a mass may be palpated, but obstruction is unusual. It is in these lesions particularly, of course, that a diagnosis of appendicitis is made because of right lower quadrant pain. This symptom, accompanied as it is in 50 per cent of cases by leukocytosis, makes a diagnosis of appendicitis most likely.[81]

The next most common symptom is increasing constipation. Loss of weight, palpable mass and bloody stools occurred next in order of frequency. In lesions of the left half of the colon, which accommodates bulky material and has a smaller lumen, obstruction is more frequent and anemia not seen as often. Pain is early and usually generalized. Increasing constipation is common, alternating with diarrhea. The rectum is the most frequent site of bowel cancer in the young. It is usually explained on the basis of irritation because of stagnation and stasis. The greatest number (79 in Bonelli's series) occurred in the 11 to 15 year age group, though others have occurred as young as two and one-half and on up to 20.

Early, these cancers are silent, but can be found easily on rectal examination. In Mayo and Madding's[54] series the diagnosis was made in 100 per cent of cases by digital and proctoscopic examination alone. Any new symptom such as change of bowel habit or rectal bleeding demands investigation by digital or proctoscopic examination.

Treatment

Radical surgery is indicated as in adults. In all these cancers early treatment is the only hope, since the malignancy progresses at a rapid rate in the young. Those we have reported were often found unexpectedly at operation, sometimes not until autopsy. Barium enema

and endoscopic examinations are difficult procedures to put a child through, but bearing in mind the indications mentioned here, they should be utilized in case of obscure and persistent symptoms related to the large bowel and rectum.

Prognosis

The prognosis in these children is extremely poor. The rate of growth in the young is rapid, and because the condition is seldom suspected it is found too late. Williams, in discussing cancer of the colon in children, says that the longest recorded survival time was four years postoperatively. In Bacon's series the average survival time, postoperatively, was ten months.

Summary

Cancer of the large bowel and rectum does occur in children, though rather rarely. Because its spread is rapid in children, it behooves us to find it at the earliest possible time if we hope to arrest the disease. This can be done only by being aware of the possibility and keeping on the alert for it.

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4614 Sunset Boulevard
Los Angeles 27, California (Dr. W. H. Snyder, Jr.)

ANORECTAL DISEASE

CLAUDE G. MENTZER, M.D.

In order to obtain a general idea of the relative frequency of anorectal inflammatory disease as compared with other proctologic disorders, a review of the author's 147 consecutive

TABLE 1. *Summary of Findings in 179 Consecutive Pediatric Proctologic Cases**

DIAGNOSIS	
Fissure (9 with cryptitis)	47
Abscess and fistula	12
Prolapse or procidentia	13
Cryptitis (1 with papillitis)	5
Hemorrhoids (all external)	4
	81
Constipation with negative findings	21
Polyps	7
Fecal impaction	6
Perianal erosion	4
Congenital anomalies	4
Parasites (pinworms)	4
Ulcerative colitis	3
Proctitis	3
Sebaceous cysts (perianal)	3
Intussusception	5
Foreign bodies	3
Hemangioma	1
Impaling	1
Criminal attack	1
	147
Negative	32
Total	179

* Includes series reported in 1947.[5]

cases is shown in Table 1. Table 2 shows the comparative frequency of these diseases reported by different proctologists.

TABLE 2. *Comparative Frequency of Anorectal Diseases as Reported by Various Authors*

AUTHOR	NO. OF CASES	FISSURE	FISTULA & ABSCESS	PROLAPSE OR PROCI-DENTIA	CRYPTITIS & PAPILLITIS	HEMOR-RHOIDS
1923 Gant[3]	263	25	19	—	—	17
1947 Mentzer[5]	147	47	12	13	5	4
1947 Murray[6]	100	38	8	18	—	—
1950 Schapiro[9]	2700	129	53	83	12	25

INCIDENCE

It will be noted that the anatomic age of the child has an influence on his predisposition to specific anorectal diseases. Schapiro[9] gives a workable classification of these anatomic ages:

Newborn (age A): birth through the third month
Infant (age B): fourth month through the second year
Childhood (age C): third year through the fifteenth year.

The initial cause of anorectal disease is the trauma produced either by the mechanical abrasion of the lining of the anal canal caused by a hard stool or foreign body, or by the irritating action of highly acid diarrheal stools. Through these abrasions the infecting bacteria of the intestinal content may gain entrance into the deeper perianal structures.

The histories of the first 81 patients in Table 1 showed that constipation was the most common disorder, having occurred in 21 normal patients, in 18 of the 47 fissure, four of the 12 abscess-fistula, and five of the 12 prolapse or procidentia cases.

Diarrhea was the next most frequent offender, having occurred in five fissure, four abscess-fistula and three prolapse or procidentia cases.

The chain of events which causes the various disease processes in the anorectal area is clearly and briefly stated by Nesselrod[7] (Fig. 36):

STAGE I. The anal crypt acts as a funnel through which infectious material from the stool, especially soft or liquid stool, is guided into anal ducts and glands.

STAGE II relates to invasion of perianal and perirectal tissues by infectious material either directly through the rupture in the gland duct structures or indirectly by way of the blood vessels and lymphatics.

These first two stages of anal infection transpire without clinical manifestation.

STAGE III is concerned with the clinical manifestations. It is the clinical manifestations which bring the patient to the physician.

Anal infection, once it has invaded the perianal glandular tissue, spreads along lines of least resistance. The direction in which the infection spreads and the different structures which it invades determine what anal disease will be produced; the local and general resistance of the patient to the infection determines its severity (Fig. 36).

Anorectal disease in the pediatric age is also influenced by a specific

anatomic trait peculiar to the infant, namely, the almost vertical position of the straight sacrum and coccyx and the insecurely attached, straight rectum-anal canal tube (Fig. 37, A). These characteristics appear to play a deciding role in the lateral distribution of fissures and fistulas and in the occurrence of procidentia in the age B group.

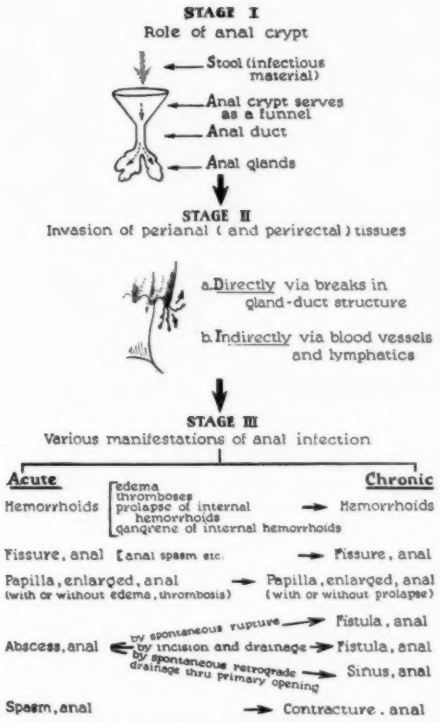
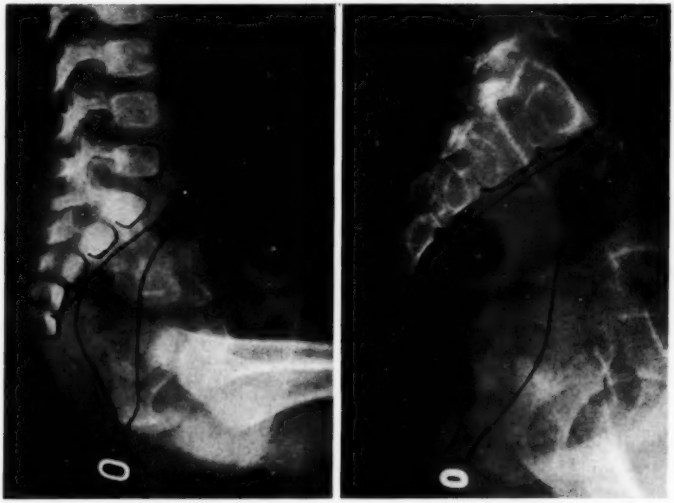


Fig. 36. Diagrammatic representation of anal infection. (Nesselrod: Proctology in General Practice.)

As the infant grows into childhood, the sacrum and rectum relation changes to assume the adult pattern. The sacrum develops a posterior concavity, and the coccyx is drawn forward by the developing levator ani muscles. The straight rectum-anal canal tube then assumes an almost right-angled relation of the rectum to the anal canal (Fig. 37, *B*). This anatomic development gives better support to the rectum and directs the area of trauma by the oncoming stool to a point preponderantly in the posterior arc of the anal canal. This explains why procidentia occurs less frequently as the child grows older, and why

fissures and fistulas tend to occur more frequently in the posterior position as in the adult.



A B

Fig. 37. Differences in the sacrococcygeal curve and the anorectal contour at different ages. A, Shallow sacral curve and almost straight anorectum in a 1½ year old child. The coccyx is not visible. B, Deep sacral curve and a right-angle relation of rectum to anal canal in a 9 year old child.

GENERAL SYMPTOMS

Characteristic prodromal symptoms are tenesmus, colic, irritability and reverse peristalsis (loss of appetite, nausea and vomiting). These symptoms are caused by the stimulation of the cerebrospinal and autonomic nerves which meet at the pectinate line, and may be due to hard or diarrheal stools or to anorectal infection.

The two most frequent symptoms are pain and bleeding. Anal fissure and, occasionally, cryptitis may cause pain with and after defecation. In perianal and perirectal abscesses the pain is continuous and throbbing.

Bleeding is the next most common symptom.

HEMORRHOIDS

Hemorrhoids in the pediatric patient are mentioned only to emphasize some common misconceptions about them. Bleeding internal hemorrhoids are rare at this age. Frequent causes of bleeding from the anus in an otherwise healthy child are a polyp, intussusception and,

occasionally, an anal fissure. There is another source of error: the rectal mucous membrane of an infant, which is redder than that of the adult, is frequently mistaken for hemorrhoids. This redness is further deepened by the congestion of the mucous membrane caused by the straining at stool, which may, in turn, produce a slight prolapse, often mistaken for a hemorrhoid.

The various factors which tend to produce hemorrhoids must operate over a long time. Some of these factors are the upright stature of man, infection in the perivascular structures, poor vein support, and lack of valves in the hemorrhoidal veins. Reuther,[8] however, demonstrated that there are valves in the middle and inferior hemorrhoidal veins in the fetus and infant, which later become nonfunctioning as the veins dilate and the valves atrophy.

Thrombosed hemorrhoids (perianal hematomas) were reported in four cases in this series (Table 1). The treatment is the same as that for an adult. If the hemorrhoids persist over a week or 10 days and the patient has continued pain which is not lessened by hot sitz baths, removal of the hematoma is in order.

PROLAPSE AND PROCIDENTIA

Prolapse and procidentia, though not uncommon in the infant (Table 1), should not be neglected even though later in childhood these conditions may improve or disappear. In this series there were 12 prolapse or procidentia cases, two in age A, six in age B, and four in age C. Both conditions are amenable to treatment.

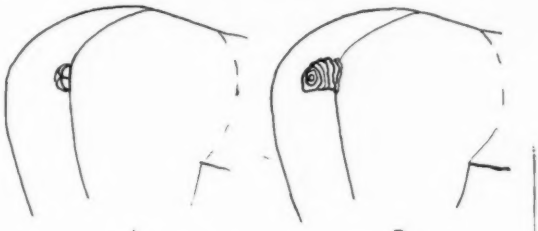


Fig. 38. *A*, Prolapse; *B*, procidentia.

Prolapse involves only the mucosal and submucosal layers, while procidentia includes all coats of the rectal wall in its protruding mass. The former presents radial folds of mucosa protruding through the anus, while the latter presents a mass which has circular rugal folds of mucosa parallel to the anal surface caused by the contraction of the circular muscle fibers of the rectum (Fig. 38).

Procidentia must be differentiated from sigmoid intussusception.

Both give rise to rectal bleeding mixed with mucus. Sigmoid intussusception, however, does not often protrude from the anus, but can be felt digitally as a cervix-like mass in the rectal ampulla. When it does protrude, the examining finger can be inserted between the sphincter and the protruding mass, but not with procidentia.[4, 13]

Causes of Procidentia

With the exception of the relatively straight sacral curve in the infant, the causes of procidentia in the young and the old are similar. In the infant the weakness of the pelvic floor is due to the incompletely developed anal and levator musculature and fascial attachments of the rectum. In the aged this weakness is due to the atrophy or wasting away of these same muscular and fascial supports. In both, the procidentia is more likely to occur and to be made worse by such contributing factors as malnutrition, straining at stool associated with diarrhea or constipation, the downward peristaltic tug caused by the presence of a polyp, coughing or crying.

Treatment

Prolapse. Terrell[11] states: "Treatment should begin as soon as the patient presents himself and the diagnosis is made. Each time prolapse occurs, it stretches the sphincter muscle and the attachments to the pelvis and other supporting structures so that with each succeeding bowel movement the prolapse occurs with greater ease. The objective of therapy is to retain the prolapse in its normal position by the artificial creation of a supporting fibrosis in and about the rectum."

The treatment of simple prolapse is not complicated. Submucosal injections of a sclerosing agent similar to that used in the treatment of internal hemorrhoids will correct the condition. The agents most commonly used are 5 per cent quinine urea hydrochloride or 5 per cent phenol in oil.

Procidentia. The methods of treatment of procidentia are many and varied, and each appears effective in different hands. The aim of all treatment is to produce adequate fixation of the rectum in its normal location. The success of treatment also depends upon the removal of all etiologic factors, so that the rectum will remain above the anal sphincter when the procidentia is reduced.

Other factors which tend to aid in the cure are the edema and the inflammatory reactions incident to the procidentia. When the protruding mass is reduced, the reaction in the tissues tends to produce scar tissue which helps fix the rectum in place. This action is further abetted by the injection of a sclerosing solution.

Terrell,[11] Tordoir[12] and the author[5] have obtained excellent results with 5 per cent quinine urea hydrochloride. Terrell[11] describes part of his treatment as follows.

> A 2-inch needle is inserted through the mucosa, just above the pectinate line, and pushed vertically upward submucosally for a distance of one to 2 inches. As the needle is withdrawn, 0.25 to 0.5 cc. of the solution is injected evenly. The injection is made in each of the four quadrants. The same procedure may be repeated at weekly intervals. These injections will stiffen and thicken the rectal wall. If this treatment is not sufficient, the author injects 5 per cent phenol in oil, 1 to 2 cc. submucosally, in two opposite quadrants above the area of quinine injection and in the other two quadrants a week later.

Better results will be secured if the bowels are well cleaned out before treatment. After treatment a fair-sized piece of cotton is placed against the anus, and the buttocks are strapped together with adhesive. Adequate sedation is given to keep the patient asleep over night. If possible, defecation is delayed for 24 hours after treatment. A 2- to 4-ounce water enema is given to produce easy defecation.

In five cases the author has had excellent results with a solution of 1:2000 hydrochloric acid injected perirectally as described by Smith.[10] This method is not without hazards and requires hospitalization and adequate anesthesia, whereas the method described by Terrell does not.

Turell,[14, 15] Tordoir,[12] Gabriel[2] and Terrell[11] have found the Thiersch method effective. This procedure mechanically keeps the procidentia reduced by the use of a silver wire placed as a purse string about the anal sphincter. Turell arbitrarily keeps the wire in place six weeks, although he suggests that this length of time is probably longer than necessary.

CRYPTITIS AND PAPILLITIS

Infection of anal crypts and the associated hypertrophied papillae is not as common in infants as in adults. Crypts were associated with 12 of the 47 fissures in this series and with seven of the 12 abscess-fistulas.

Crypts are frequently overlooked not only because a proctoscopic examination is not always a routine part of a complete physical examination, but also because many physicians fail to use the hooked probe, with the aid of which crypts may be demonstrated. Only a scarred crypt in the chronic stage may be palpated. Once the crypts and anal ducts and glands become infected, the direction of spread determines whether a fissure or an abscess-fistula will result.

ANAL FISSURE

Anal fissure is by far the most common anorectal disease in infancy and childhood, accounting for 47 of the 147 patients in this series

(Tables 1 and 2). Most anal fissures occur in the age B group. Anal fissure is a disease of many facets, and in this age group differs in both location and treatment from fissure in the adult patient. Medical or conservative management was sufficient to cure the majority of fissures in these juvenile patients, whereas surgical intervention is the rule for adults.

The predominant symptom was pain, which was present in 44 of the 47 patients; 19 had bleeding at some time during the course of the disease, 20 were constipated, five had diarrhea, and 12 had anal crypts. Most of these patients had more than one fissure, located more frequently on the lateral than on the posterior or anterior aspect of the anal canal.

Pathogenesis and Age Incidence

Anal fissure occurred rarely in the age A group, having been present in only five cases. The greatest number of anal fissures, 34, was found

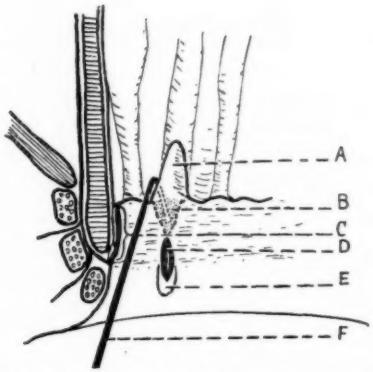


Fig. 39. Fissure quadrad. *A*, Anal papilla; *B*, burrowing crypt; *C*, pecten band; *D*, anal fissure; *E*, sentinel pile; *F*, hooked probe.

in the age B group. Almost invariably the fissures occurred on the lateral rather than the posterior or anterior wall and were often multiple. The 10 anal fissures in age C group followed the pattern of fissures in the adult; that is, they were usually single and most often located on the posterior wall.

The point of greatest trauma by the oncoming stool is changed by the anatomic alterations of the vertical sacrum-coccyx and straight rectum-anal canal relation in the age B group to the right-angled relation of the anal canal and rectum in age C (Fig. 37). The traumatic factors are then greatest posteriorly and account for the posterior location of anal fissures as the child grows older.

Fissures in the age B group, 34 of 47 in this series, were situated laterally, had little crypt involvement, and most frequently followed the passage of a large hard stool. Five of the fissures followed diarrhea.

Fissure in the older child, age C group, follows the pattern of fissure in the adult. After the entrance of infection into the anal crypt and duct and anal gland the adjacent papilla hypertrophies, and there is an attempt at healing of the accompanying burrowing cryptitis. If the process becomes chronic, inelastic scar tissue is laid down as part of the reparative process. Trauma produced by the passage of a large stool often causes a tear in the skin and subcutaneous tissues in the scarred area. At the lower end of the fissure a so-called sentinel pile develops. The development of cryptitis, papillitis, fissure and sentinel pile, in sequence, has been dubbed "fissure quadrad" (Fig. 39).

Treatment

Many anal fissures in the age A and B groups are cured by dilation of the anal sphincter incident to proctoscopic examination and/or by the correction of the constipation or diarrhea. Terrell[11] states that he has never had to resort to surgical intervention in this age group. However, 17 of my 47 patients with fissure were operated upon. The surgical cases were equally divided between the age B and C groups.

Medical management is as follows: (1) At bedtime the baby is placed on its abdomen and one ounce of mineral oil is injected into the rectum (using a 1-ounce ear syringe); the patient is kept in this position long enough to permit the defecatory impulse to stop. (2) When the baby awakens in the morning, and before defecation, a small amount of an anesthetic ointment is placed on the anal verge and is rubbed into the anal fissure. (3) If the baby has no bowel movement after the first feeding, an enema containing 2 to 4 ounces of warm water is given to establish a habit time and to soften the stool. (4) After the defecation the baby's buttocks are "dunked" into a pan of warm water for 10 minutes. (5) An ointment containing a broad-spectrum antibiotic is rubbed into the fissured area after the sitz bath. (6) Occasionally the mother is shown how to dilate the anal sphincter digitally. In my experience this regimen cures almost all fissures occurring in this age group unless the fissure is accompanied by a scarred anal crypt.

When the fissure quadrad is present in the age C patient, definitive surgery is indicated for cure. Either drop ether or Vinethene or a combination of these anesthetic agents is used. Infiltration anesthesia, using a 1 per cent procaine hydrochloride solution, helps to eliminate deep inhalation anesthesia. This is combined with an injection of 5 cc. of an oil-soluble anesthetic solution deep into the posterior aspect of the

anus and the area of the fissure. The anal sphincter muscle is then gradually dilated to admit two fingers. The surgical procedure consists in excision of the fissure-bearing area along with the offending crypt, papilla and sentinel pile. If this procedure is followed, the fissure will not recur. Postoperative care is the same as for medical management of fissure.

ANAL ABSCESS AND FISTULA

Anal abscesses and fistulas are not uncommon in juvenile patients (Table 1). In this series all fistulas in the age B group had their origin in lateral or anterolateral crypts. A recent report by Venturo[17] of seven patients in this age group showed the same distribution of fistulas. This distribution is of particular interest because the majority of fistulas in adults have their origin in a posterior anal crypt. After crypt infection an acute abscess develops. When the abscess is incised or there is spontaneous rupture, the contracting abscess cavity becomes a fistula or a chronic abscess. The treatment is always surgical and in no way differs from that accorded adults.

Symptoms

The case history of one patient in this series demonstrates the symptomatology well.

J. S., a white boy, 6 months old, began to cry on defecation at the age of 10 weeks. The mother noticed a lump on the right side of the anus which ruptured spontaneously with subsequent periodic drainage of pus. During the first few weeks after birth he had had a severe diarrhea, but after the cessation of the diarrhea he was constipated up to the time of examination. He seemed irritable, whimpered, never smiled, ate and slept poorly, and never attempted to sit. A physical examination revealed a poorly nourished, moronic-appearing baby, with 2 complete intersphincteric anal fistulas, one on each lateral side. One month after bilateral fistulectomy he had made a good recovery, with a disappearance of all the abnormal symptoms.

In the group of 12 cases (Table 1) there were three abscesses without demonstrable primary crypt origin. One was a congenital external perianal sinus. There were eight complete fistulas which originated in crypt infections. All were lateral or anterolateral in position and directed radially in a straight line from the involved crypt to the external opening. (The previous bowel habits in the eight patients were equally divided between constipation and diarrhea.) With the exception of one, all fistulas were perianal or low ischio-anal in location and reached the perianal space through the body of the subcutaneous part of the external sphincter muscle or between the external sphincter and the internal sphincter muscles. The eighth patient had a massive abscess which almost filled the left ischio-anal space and penetrated the levator ani muscle to involve the supralevator space.

Pathogenesis

In reviewing the three stages of anal infection, and specifically the third stage (Fig. 36), it is apparent that once the infection has passed the barrier of anal crypt, anal duct and anal gland, it spreads via lymphatic and blood routes or by direct extension along lines of least resistance. In this area the structures along which infection can spread most easily are (1) the terminal fibromuscular extensions of the longitudinal muscle which separate the various portions of the external sphincter muscle, (2) the loose perivascular connective tissue stroma,

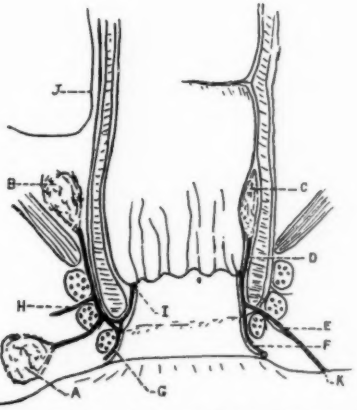


Fig. 40. Abscess-fistula. *A*, Ischio-anal abscess; *B*, supralevator abscess; *C*, submucous abscess; *D*, submucous fistula; *E*, complete intersphincteric anal fistula; *F*, complete subcutaneous anal fistula; *G*, incomplete subcutaneous anal fistula; *H*, intersphincteric anal fistula between deep and superficial portions of the external opening in a crypt; *I*, internal or primary fistulous opening in a crypt; *J*, peritoneum; *K*, external or secondary opening of fistula in the perianal skin. (Jackson Memorial Hospital Bulletin, July, 1939, Miami, Florida.)

and (3) the perianal fat, which offers little resistance to infection. Figure 40 shows the various possible abscesses and fistulas which may form. The most common in this series was the intersphincteric anal fistula.

The peculiar anatomic trait in the infant, namely, the relatively straight vertical rectum-anal canal, explains why the effects of trauma are not primarily on the posterior anal wall as in the adult (Fig. 37). The predilection of the lateral crypts for trauma and the entrance of infection in the infant has not been satisfactorily explained. Nevertheless, this is the site of most frequent involvement of both fistulas and fissures in the age B group.

Treatment

When the perianal or ischio-anal abscess ruptures or is incised, an external sinus is formed. If the primary opening in the anal crypt had been patent, the abscess would gradually have drained into the rectum with minimal symptoms to the patient and would have constituted an internal sinus.

The basic concepts of proctologic practice probably are violated more frequently in the treatment of abscess about the anus and rectum than in any other disease in this area. First, penicillin is given indiscriminately "to see if the abscess can be aborted." If an antibiotic is to be given, one of the broad-spectrum variety should be prescribed to take care of the systemic spread of the infection. Second, Terrell[11] states that an abscess must be suspected when the patient has had pain for over 36 hours, and that it is mandatory to prove its presence or absence. Third, the most serious "don't" in proctology is to wait for the abscess to "point," because it may spread in many directions instead of toward the skin. Therefore, if an abscess is present, immediate incision and drainage is mandatory no matter what its depth. It must also be remembered that mere incision of a perianal abscess does not always prevent fistula formation.

Aderne,[1] 500 years ago, stated: "If a fistula is in a chronic state, represented by periodic drainage and a cord-like structure with a primary and secondary opening, surgical opening of this tract from one end to the other is necessary no matter how much muscle has to be cut in order to effect a cure."

There are three schools of thought in the surgical treatment of fistulas. They are (1) fistulotomy, (2) fistulectomy, and (3) a combination of the two methods. Fistulotomy usually cures and is probably done most frequently for the pediatric patient. I prefer fistulectomy in that part of the fistula outside the anal sphincter and fistulotomy through the muscle portion. The fistulotomy portion of the operation prevents the retraction of the muscle fibers which are adherent to the base of the fistula.

Turell[16] feels that fistulectomy, by disposing of all infected scar tissue, is a more complete operation that eliminates the possibility of the development of malignant degeneration in the unexcised portion of the fistula many years after the primary operation.

SUMMARY AND CONCLUSION

Anorectal disease in the pediatric age is most prevalent in the group aged four months through the second year.

The most common proctologic diseases are anal fissure, abscess-fistula and prolapse or procidentia, in that order of frequency. Hemor-

rhoids and papillitis are rare. Cryptitis is of concern only when it is associated with fissures and/or fistulas.

A common anatomic trait, the relatively straight sacrum and coccyx and the insecurely attached, straight rectum-anal canal tube, gives rise to laterally placed fissures and fistulas in the infant. This same trait offers less support to the downward tug of the rectum during defecation, so that procidentia is not uncommon.

The significant part played in anorectal disease by the trauma of a constipated or diarrheal stool has been described.

Treatment of proctologic disorders should start as soon as a diagnosis is made. In cases of abscess and fistula treatment is always surgical. In fissures and procidentia treatment may be medical or surgical or a combination of both.

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434 N. E. 95th Street
Miami 38, Florida

CONSTIPATION IN CHILDREN

HARRY BAKWIN, M.D.

By constipation is meant infrequent or difficult evacuation of the feces. It is a common complaint in children and is generally of minor importance. It may, however, become the focus around which major parent-child conflict revolves.

NORMAL STOOL INFREQUENCY IN INFANTS

The stools are generally less frequent and firmer in bottle- than in breast-fed babies, owing, probably, to the higher calcium content of cow's milk. Boiling the milk seems to have a constipating effect. Infants not infrequently go for 48 hours or even longer without a stool. This is generally unassociated with any discomfort, but some infants, after three or four days without a stool, become irritable, restless or listless. The symptoms disappear promptly after emptying of the distended rectum. A two year old girl screamed when she sat up, but seemed all right when she was lying down. Her rectum was found to be filled with hard feces (first revealed by the rectal thermometer). The postural discomfort from which, presumably, she was suffering subsided at once when the rectum was emptied.

Infants may be safely permitted to go for 48 hours without a stool. If the parents are concerned, an occasional teaspoonful of milk of magnesia will relieve the symptom. Milk of magnesia probably acts by drawing water into the colon and by stimulating peristalsis. Its action is brief. It is preferable to the widely used prune juice. Prune juice has a cathartic effect, but the optimal amount is sometimes difficult to measure, and diarrhea may result. Moreover, some children

From the Department of Pediatrics, College of Medicine, New York University-Bellevue Medical Center, New York City.

do not like prune juice. Using brown sugar as the added carbohydrate in the formula is generally effective in overcoming stool infrequency in infants.

As far as is known, stool infrequency in infants is not related to constipation in later life unless the use of cathartics, suppositories or enemas becomes habitual. With the introduction of foods which have more residue the stool infrequency ordinarily corrects itself.

PSYCHOGENIC CONSTIPATION

The defecation reflex in infants is simple and direct. In the normal course of events the contents of the colon are conveyed to the pelvic colon by mass peristaltic movements. These take place several times a day, usually in response to the entrance of food into the stomach (the gastrocolic reflex). The fecal mass remains in the pelvic colon until a mass peristaltic wave forces the material into the rectum, and the call to defecation occurs. Expulsion of the stool is effected by relaxation of the anal sphincter and the muscles of the pelvic floor, contraction of the muscles of the abdominal wall, diaphragm and back, drawing up of the legs, and forceful expiration with closure of the glottis. Under normal circumstances the rectum contains a small amount of fecal material.

At first the reflex is unaffected by extraneous factors, the stool being passed without regard to time or place or other circumstances; but, as the child grows older, conditioning influences become more and more important. As part of the process of toilet training he learns that defecation can be suppressed at will. Later he comes to appreciate that there are circumstances under which social usage demands that he retain his stool, often for several hours, and the number of inhibiting influences multiplies. In certain households where the mother is overfastidious the child may be discouraged from having a stool anywhere except at home. In many adults, less often in children, defecation is highly conditional and takes place only at a particular time of the day, in a specific place, after certain foods or drinks, and so on.

ETIOLOGY

Failure to respond to the call to defecation is a common cause of constipation in children. As the fecal mass remains in the rectum, the urgency of the reflex diminishes, and the stools become hard and more difficult to pass. Sometimes fissures appear which make passage of the stool painful.

The use of cathartics, by emptying the bowel at unfamiliar times and at irregular intervals, further interferes with conditioning. Strong

cathartics empty the bowel so completely that more than one day is necessary to fill it. Irritation of the colon by cathartics sometimes leads to spasm of the left colon which is visible with the proctoscope. Colonic spasm may also be produced by the tension accompanying the constipation.

In many children with constipation the mother is herself constipated. She transfers her own feelings of discomfort to the child and is consequently overeager to see that the youngster is protected from her own sufferings. Her excessive interest and zeal, often accompanied by coercive training methods, may rouse the child's negativism and make him resistant to going to the toilet when he needs to. He may become tense about the toilet situation, and in this way spasm of the colon becomes an added factor in his constipation. The possibility of a hereditary factor cannot be ruled out.

The following improper training procedures associated with constipation have been encountered by Huschka:[6] too early training; shaming or punishing for failure to comply; the overuse of suppositories; sending the child to the toilet too often and keeping him there too long; rigidity of the toilet schedule; putting a high premium upon perfect performance; making the child express his wants in speech before helping him to the toilet; strapping the child to the toilet seat until he has had a bowel movement.

Prugh[9] found that colonic dysfunction (constipation with or without fecal incontinence) was twice as frequent in a group of children with early bowel training as in a group of children trained at the usual time. This finding suggests that early training may be a factor in the bowel difficulties of children, but the groups were not large enough to yield statistical reliability. Moreover, no close relationship was apparent when the type of bowel training and colonic dysfunction were assessed in children completely toilet trained before one year.

Prugh concludes, on the basis of his own data and data from the literature, that the timing and nature of bowel training cannot be the sole etiologic factors in the development of colonic disorder. Mothers whose relationship to their children is close and warm can use premature and coercive toilet training methods without producing colonic symptoms. On the other hand, when strain exists between mother and child, undesirable training procedures may offer a focus around which tensions are expressed. The personality make-up of the mother and her relationship with her child are more important than the technique she applies in child rearing.

In our experience psychogenic constipation occurs not infrequently in children in whom a history of premature or coercive toilet training procedures cannot be obtained. In a certain number of constipated and incontinent children training has been overpermissive. If one waits

too long and fails to institute training procedures when children are developmentally ready to be trained, they may acquire absorbing play interests from which they refuse to be separated in order to be put on the toilet.

Readiness for toilet training implies the ability to sit firmly on the toilet seat, sufficient control so that the child can hold the stool until he is put on the receptacle and can then release it, some means of communicating his discomfort to the mother, and some understanding of the social implications of the act. Naturally the infant does not mature in all these phases at the same time, but it seems reasonable to begin toilet training before all these developmental stages have been reached. For example, it would appear that the ability to retain the stool is acquired earlier than the ability to release it. Consequently many young children wait until they are placed on the receptacle, but are unable to release it there. When they do have a stool shortly after removal from the pot, mothers frequently get the impression that the youngsters are being spiteful.

Toilet training is usually fairly complete by 15 to 18 months. The world of the preschool child is an exciting place. During this age period he discovers the nature of the material world around him; objects, colors, textures, dimensions begin to take on meaning. If the use of the pot is not established as a habit by 18 months or so, a good many children resist withdrawal from their interesting explorations, and this may be the beginning of a conflict around bowel function.

A considerable number of constipated children do not impress one as emotionally disturbed, the bowel difficulty being an isolated symptom. In those who are emotionally disturbed it is not always easy to tell whether the bowel difficulty caused the emotional upset or whether the emotional upset caused the constipation. It is conceivable that, in some of these children at least, the basis is an anatomic abnormality somewhere in the lower bowel or in its innervation. The fact that children outgrow their bowel difficulties does not negate this hypothesis, since other developmental abnormalities, e.g., the language difficulties and enuresis, are also outgrown.

A certain number of overprotected children refuse to chew and swallow coarse foods. Consequently there is lack of sufficient intestinal residue which aids evacuation of the bowel. Milk is a constipating food, probably owing to its low residue and its high content of calcium.

Many children fail to respond to the call because they are too busy playing or watching television or listening to the radio. Others are so anxious to get to school in the morning that they do not take time to go to the toilet before leaving home. By the time they get to school the call may have passed, or the child is hesitant to ask for permission to leave the classroom, or the teacher will not allow him to go.

Girls are more frequently constipated than boys. On the other hand, bowel training problems, such as resistance to training and soiling, are twice as common in boys.

SYMPTOMS

In most cases of psychogenic constipation general symptoms are lacking. There may be abdominal distention and discomfort, restlessness or apathy, chilly sensations and headache. These symptoms are due, for the most part, to mechanical distention of the rectum by impacted feces rather than to absorption of "toxins," since relief of symptoms follows promptly after emptying the rectum. Moreover, the symptoms have been reproduced by filling the rectum with an inert substance. The feces can usually be felt through the abdominal wall in the left lower quadrant as hard, irregular masses.

Occasionally fissures and hemorrhoids may result from the constipation. They may also cause it.

Fecal incontinence frequently accompanies severe constipation. The rectum is filled with a firm, irregular mass of stool, bits of which are passed into the clothes from time to time. In addition, small amounts of greenish, mucoid, fetid material, which are produced by irritation of the rectal wall, may be passed several times a day, and "diarrhea" may be the presenting complaint (obstipatio paradoxica).

In some children the constipation manifests itself by only occasional episodes of fecal incontinence.

Such was the case of M.E., a girl 5½ years old, who was seen because of soiling. The stools were normal in consistency, sometimes greenish in color. They were passed frequently, usually a small amount at a time. Bowel training had proceeded normally, and there had been no previous difficulty except a tendency to stool infrequency. On digital examination the rectum was found to be filled with a firm, irregular mass of feces. The rectum was emptied with enemas. The child remained well for several months, and then a second, similar episode occurred. It was treated in the same way. The girl, now 14 years old, has been free of these episodes ever since. She states that she uses the toilet about once a week. She is a bright, attractive child with many interests. Her mother believes that she does not want to take time to go to the toilet. There are no symptoms. The parents are not concerned about the constipation.

Richmond, Eddy and Garrard[9] studied 10 children with functional megacolon and encopresis. They found that the majority of children in their group were unusually obedient and conforming and overneat in everything except their bowel training. The children performed well at school and were often favorites of their teachers. They seemed to control their hostility and aggressiveness and impressed adults favorably. On the whole they related poorly to their own age group, and their attitudes were stoical with infrequent displays of overt emotion. The majority were strikingly immature in appearance. The mothers of

the patients seemed incapable of expressing genuine warmth or feeling.

The following features serve to differentiate psychogenic megacolon from congenital aganglionic megacolon:[9] In severe cases of psychogenic megacolon soiling is the major complaint. The rectum is packed with stool which may be firm and may require strenuous efforts to empty. After evacuation the rectum remains widely dilated. Other features are an onset after neuromuscular control is expected, i.e., about two years or later, in contrast to aganglionic megacolon, which usually manifests itself in the newborn period; defecation in the standing or supine position rather than while seated on the toilet; holding the stools for days or even weeks, with periodic voluminous stools passed spontaneously or after enemas; absence of a "normal" segment of rectum or rectosigmoid in the roentgenogram; and a benign course.

SPASTIC CONSTIPATION

Constipation may be produced by spastic narrowing of the sigmoid, thereby preventing the stool from passing into the rectum. The spasm is sometimes due to inflammation or an infected tumor, but it occurs much more often in anatomically normal colons as a response to emotional tensions.

The frequency of this form of constipation in childhood has not been studied. It probably occurs earlier and more commonly than is generally believed. Davidson[4] has seen stool holding in an infant 10 months old. In his experience diarrhea during the second year of life is often preceded by constipation in infancy. Almy[1, 2] has seen a child of one and one-half years with spastic constipation. The condition disappeared when parent-child tensions were relieved.

The stools consist of small, round, firm masses or are ribbon-like or pencil-like. Mucus may be present. The delay in the passage of stools in these patients takes place at the sigmoid or higher up in the intestinal tract. The rectum is empty or contains only a few firm pellets of feces.

Changes in the shape and motility of the colon have been induced by Almy, Kern and Tulin[2] with a number of stressful stimuli. They used immersion of the hand in ice cold water, induction of headache by a tight band around the head, insulin-produced hypoglycemia, and discussion with the patient of stress-producing life situations.

With all these methods sustained contractions of the colon took place, although the changes were not necessarily related to the intensity of the stimuli. However, when colonic changes were observed they were uniformly associated with emotional tension and bodily reactions designed to protect the individual when his security is threatened.

These changes in the colon following stressful conditions were observed in normal persons as well as in patients suffering from spastic constipation. The authors regard the fundamental difference in the patient with spastic constipation to be, not the behavior of the colon in response to stress, but his greater susceptibility to stress-producing situations.

The fact that increased activity of the colon produced constipation rather than diarrhea is explained by the observation that these contractions are nonpropulsive.

TREATMENT

The first effort in the treatment of children with psychogenic constipation should be directed toward discovering circumstances in the environment which may be disturbing the child. Allowing the parents to talk freely, with special attention to attitudes toward bowel habits, will usually give the physician clues to the source of the difficulty and will point the way toward correction. The relief which the parents get from free discussion, with an occasional suggestion from the physician, will often have a remarkable effect on lightening the tensions in the home.

Discussion with the parents should be designed specifically to relieve concern about the bowel function. The parents need to know that a daily stool is by no means essential, that many quite normal persons get along with a stool every second or third day or even less frequently; and that "toxins" are not absorbed from the retained stool. Parental inquiries, urgings, pleadings, exhortations and commands to go to the toilet should be discontinued. The use of drastic cathartics, frequent enemas and suppositories should be stopped.

An important part of the treatment is establishment of a regular routine. From the time when the youngster is trained to the toilet he should have a definite time for evacuation—preferably after breakfast—and this should be adhered to more or less strictly. School children should have ample time in the morning for going to the toilet.

The drinking of excessive amounts of milk should be discouraged, since milk is a low-residue food; in addition, the large amount of calcium it contains probably has a costive effect.

Cathartics may be used during a training period. The most suitable are those which increase intestinal bulk and are nonirritating.

The efficacy of antispasmodic drugs in spastic constipation has not been demonstrated. They may interfere with the propulsive action of the bowel, thereby exaggerating the difficulty.

When fecal impactions are present, the rectal pouch must be emptied by enemas. The masses are often very hard, and mechanical

removal may be necessary. The instillation of oil sometimes serves to soften the mass and facilitates its removal.

CONGENITAL AGANGLIONIC MEGACOLON (HIRSCHSPRUNG'S DISEASE)

In congenital aganglionic megacolon obstruction to the passage of stools is due to deficiency of the parasympathetic ganglia in a portion of the rectum or colon. Since this part of the intestinal tract is unable to sustain effective peristaltic waves, the stool accumulates proximal to the lesion and the colon dilates and hypertrophies. If impaction of feces is allowed to persist, necrosis, infection, ulceration and perforation may follow. The mortality has been variously estimated at 10 to 25 per cent. The ganglion deficiency is usually in the lower rectum and sigmoid, but occasionally is higher up in the large intestine.

Symptoms

In cases of congenital aganglionic megacolon the symptoms of intestinal obstruction appear within the first few days of life in 80 per cent or more of the cases. The syndrome manifests itself at this age by constipation, abdominal distention and vomiting. In some infants no meconium whatsoever is passed spontaneously; in others it may be scant or unusually firm; still others have a normal meconial discharge. Abdominal distention is a regular accompaniment of the constipation. It is progressive and often of alarming dimensions. Vomiting takes place in about half of the cases. It is usually frequent and large in amount. Despite these symptoms the general condition remains good.

In most instances, in contrast to the other types of intestinal obstruction, the symptoms are relieved by high enema: the vomiting ceases, the abdomen assumes a normal contour, and spontaneous bowel movements appear. Then, after an interval varying from one week to two and one-half months, constipation and abdominal distention reappear.

In later childhood the picture is one of severe constipation and bouts of obstipation. There are frequent complaints of abdominal cramps, vomiting and audible abdominal gurgling. The abdominal distention varies from time to time and is often extreme. The pattern of the dilated bowel and peristalsis of the colon are often visible. Fecal masses are frequently felt, usually in the left lower quadrant. Despite this, rectal examination reveals an empty or nearly empty ampulla. The rectum is not dilated, and the wall appears normal on proctologic examination.

A familial incidence has been observed in some cases by Zuelzer and Wilson[18] and by Bodian, Carter and Ward.[3]

Diagnosis

Roentgenologic examination is a great aid in diagnosis. Barium should be allowed to run in slowly and in small amounts. For proper visualization of the rectosigmoid the patient must be examined in the oblique projection. In this position a portion of the rectum or rectosigmoid is revealed, often up to 10 cm. in length, which is consistently less than normal in caliber. The contour is frequently irregular, and often a turbulent and purposeless peristaltic activity can be seen in this area. Occasionally, a reverse peristalsis is present. As the barium is allowed to enter the dilated portion of the bowel, the involved area is obscured.

Roentgenologic examination in young infants is not always helpful, since the colon may not be distended and hence the contrast between a distended colon and a narrow lower segment may not be demonstrable. Swenson[11] has found biopsy of the rectal wall to be of help in these patients. He finds it useful also in patients with equivocal signs of the disease on radiologic examination, and in children who had a transverse colostomy early in life. This procedure should be used in children who have psychogenic constipation with great caution because of its possible psychologic effects.

Differential Diagnosis

Congenital aganglionic megacolon must be differentiated from mechanical obstructions due to anorectal malformations or cicatricial stenosis, and from psychogenic constipation. Mechanical obstruction is established by rectal examination. The differential features of psychogenic constipation are listed under that condition.

Treatment

A certain number of cases are sufficiently mild to be controlled by medical treatment. This consists of measures to assist defecation. Mild cathartics such as milk of magnesia help to keep the colonic contents soft. Enemas are necessary, the frequency depending on the comfort of the youngster. Isotonic saline solution should be used rather than tap water or soapsuds, since a considerable amount of fluid may be retained, thus diluting the body electrolytes. Sudden death during or immediately after a copious enema has been observed in these children.

In most instances surgery is necessary. This consists in resection of the aganglionic segment and anastomosis of the colon and rectum. Preliminary colostomy is generally not necessary. The over-all mortality from the operation is about 5 per cent. Relief of symptoms follows

operation except for the rare case in which resection of the aganglionic portion of the gut has been incomplete.

Operation should be deferred until such time as the aganglionic portion of the colon can be delimited in the x-ray film. This is usually not possible during early infancy. Occasionally colostomy may be necessary, especially in infants, to relieve acute obstruction.

Though the aganglionic portion of the colon is usually limited to the lower rectum, Swenson[11] has encountered cases with extensive involvement up to the transverse colon and even to the hepatic flexure. These patients have responded surprisingly well to operation. He has also seen two infants with involvement of the entire colon. Anastomosis of the terminal ileum to the anal canal, following ileostomy, yielded good results.

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132 East 71st Street
New York 21, New York

DIARRHEA IN INFANTS AND CHILDREN

SAMUEL KARELITZ, M.D.

Diarrhea is a condition in which there is an accelerated excretion of intestinal content, which, if moderate or severe, leads to incomplete digestion of food substances, diminished absorption of split foods, and excessive loss of these food elements, fluid and electrolytes.

What constitutes diarrhea is sometimes difficult to define, since one infant has one firm stool daily while another may have five to eight soft small stools, one during or after every feeding. Both infants may be well and thriving. A change in diet may increase the number and reduce the consistency of the stools of the first child, and the second may respond to the same diet with firmer and fewer stools. I have observed these differences following a change in amount or type of sugar, and often after acidification of the formula with lactic acid.

The breast-fed infant is more likely to have several soft, unformed, yellow stools with little inoffensive odor, while those of the weaned baby on cow's milk are of different color, depending on the formula, and are more formed, of greater bulk and of more distinct odor. High protein milks produce thicker stools. High fat intake produces softer stools.

As the infant becomes older there is a tendency for a reduction in frequency and an increase in size and consistency of stools. The infant or child who eats a great deal will have larger and more frequent stools.

The decision as to whether a child has diarrhea must depend on several factors: first, a noticeable or sudden increase in the number of stools, or reduction in consistency, and an increase in their fluid content, a change in color with a tendency to be green, often a change in

From the Department of Pediatrics, Mount Sinai Hospital, New York City, and the Long Island Jewish Hospital, New Hyde Park, New York.

the odor, and an increase in gas. In addition, the infant will probably have reduction of appetite, possibly nausea; he may be unhappy, cry more, and be restless, and possibly distended. In general he will look sick, or at least not as well as before diarrhea started.

Diarrhea is important at any age, but it is most significant in the first year of life, especially in the first few months. In the following discussion I shall be mainly concerned with diarrhea in the newborn and in the child through the first few years, and shall comment briefly on diarrheal conditions in older children.

INCIDENCE AND MORTALITY

Diarrhea was at one time one of the leading causes of death in the first year of life. In New York City, in 1889, 31 per cent[36] of the 10,527 deaths were due to "cholera infantum." By 1938 diarrhea accounted for 8.8 per cent of 3910 deaths, and, in 1946, 4.5 per cent of 4239 deaths. A striking reduction in the incidence of diarrhea has occurred wherever pasteurization of milk has been enforced, where careful preparation and proper refrigeration of milk are widely practiced, and where good hygiene has reduced the incidence of salmonellosis and shigellosis. The extensive use of evaporated and powdered milks, which are sterile and more or less uniform in composition, has also contributed. These methods have leveled off the peak incidence of diarrheal diseases and infant mortality during the summer months.

Modern therapy of the acidosis and the circulatory collapse resulting from the diarrhea has further reduced the mortality from diarrheal diseases. In 1924 and 1925 data collected in two hospitals for the previous 10 year period indicated an average mortality of 64 per cent in one, and 63 per cent in the other. Since then, in cases of diarrhea of comparable severity, the mortality has been reduced to less than 10 per cent, and in some clinics to 5 per cent or less. This experience is not universal, since in some parts of the world severe infantile diarrhea is still a major problem and results in mortality up to 60 per cent. In our own experience we have encountered little mortality in infants over six months of age. The mortality from diarrhea is more likely to occur in the first few months of life, especially in cases of epidemic diarrhea of the newborn.

ETIOLOGY

There are many specific etiologic agents of diarrhea, the most common of which are shigellosis, salmonellosis and various types of *Escherichia coli*. Other bacteria, such as *Staphylococcus aureus*, *B. pyo-*

cyaneus, Proteus morgani, play an increased role since extensive use of antibiotics. Diarrhea occasionally occurs with typhoid. Amebic infection is not a common cause of diarrhea in infants. In recent years a great deal of information about the etiology of infantile diarrhea has been accumulated. This has been largely in connection with epidemic diarrhea of the newborn.

Epidemic Diarrhea of the Newborn

This condition, though not new, became a serious problem in many parts of the United States some 25 years ago when outbreaks in newborn nurseries were reported.[23, 45] This disease has an incubation period of two to four days, but it may extend to two weeks. Outbreaks are usually explosive and may involve 50 per cent or more of the occupants, but the average morbidity is about 25 per cent. The sick infant may show slight premonitory signs for a day, after which there follow severe diarrhea and vomiting with rapid development of dehydration, acidosis and shock. Treatment as for alimentary intoxication is of variable success. In one outbreak all may recover, while in others the mortality may reach more than 50 per cent, averaging nearer 25 per cent.[28] This difference in response to treatment is striking. In one outbreak involving 13 infants all survived.[18] In another hospital an outbreak of what seemed to be the identical disease resulted in a mortality of 45 per cent.[39] Breast-fed infants are less likely to be involved.

It was formerly thought that this infection was limited to the newborn, but it is now known that the same types of *Escherichia coli* are found in older infants and children with mild gastroenteritis.[22] Hallman,[25] Rantasalo, Tuuteri and Kotilainen studied the incidence of *Dyspepsia coli* (a name given to these strains of coli by Adam[1]) in gastroenteritis in 116 infants under one year in Helsinki between May 15 and September 30, 1952. In 100 health controls type 0-111 was found in two, both of whom had had gastroenteritis two weeks before. In the sick children 0-26 was found in 17, 0-111 in three, and 0-55 in two. Only three were breast fed, but they had supplementary feeding. None of these organisms were recovered from the nose and throat of the infants or their mothers or from other members of the family, or from the milk. In a hospital study Rantasalo[43] and Hallman found *Dyspepsia coli* in 13 per cent of the mild diarrheas, and in 56 per cent of infants with severe diarrhea type 0-26 was found most frequently, 0-11 and 0-55 less often. Neter[35] recently reported an outbreak of *Escherichia coli* 0-111 diarrheal disease in infants between three weeks and nine months of age on a surgical ward.

To date epidemic diarrhea of the newborn has been associated with

viruses, various types of salmonellae, certain strains of *Staphylococcus aureus*, group D beta hemolytic streptococcus, shigellae, and *Ps. aeruginosa*, and 12 different serologic types of Escherichia have been incriminated:[44] 0-11; B4, 0-55; B5, 0-26; B6, 0-122a; B13, 0-112a, 0-112c; B11, 0-86; B7, 0-119; B14, 0-125; B15, 0-126; B16, 0-127; B8. In an excellent review of the etiology of epidemic diarrhea Hodes[28] concluded: "The evidence summarized warrants the conclusion that certain types of Escherichia must be considered to be capable of causing infantile diarrhea along with salmonellae, shigellae and other organisms." Light and Hodes,[32, 33] Cummings[10] and Buddingh and Dodd[3] have isolated viruses from patients with epidemic diarrhea. Thus the etiology of this condition is variable. Possibly herein lies the explanation of the variability of the intensity and mortality in this disease.

Other Causes

There are many other instances of diarrhea which seem to be unrelated to specific infection; at least no specific etiologic agent has been identified.

The causes of sporadic diarrhea are multiple and varied. It may be due to an irritant. For example, castor oil[30] given to a child for constipation was followed by diarrhea which resulted in dehydration and circulatory collapse. I have observed the same sequence following the use of milk of magnesia. Dietary causes of diarrhea are numerous. A change in the composition of the infant's formula—for example, from evaporated milk to pasteurized whole milk; a change in the added carbohydrate from one sugar to another; a sudden shift from breast feeding to a cow's milk formula; the introduction of a new food—for example, orange juice—all have caused diarrhea.

Believing that raw vegetables might be preferable to cooked ones for infant feeding, I prepared carrots and string beans, finely chopped in a mechanical blendor, after scalding them in boiling water for two minutes. By error 2 ounces of this practically liquid vegetable were fed to each of several infants, and within 24 hours diarrhea followed in all. I had intended to introduce one teaspoonful of this preparation at first. Whether it was the change in consistency or some irritant in the raw vegetable which produced the diarrhea is not clear. It is not likely that infection played a role. This experience may be comparable to the "tummy ache" and diarrhea which the older child gets from eating too many green apples.

Overeating, excessive fat in the meal, highly spiced food may also cause diarrhea. It is well appreciated that the malnourished infant is more susceptible to diarrhea. Whether this is associated with a reduction in gastrointestinal enzymes or with poor absorption is not defined.

Constitutional Predisposition

The child who is allergic may have diarrhea after the ingestion of the allergenic food. For example, a child sensitive to egg, if given egg, may have intestinal spasms which are demonstrable on x-ray examination.[17] This may result in increased peristalsis and diarrhea. The child born with cystic fibrosis of the pancreas has diarrhea, probably associated with the lack of adequate enzyme activity. In the celiac syndrome diarrhea is probably associated with the poor absorption of fat and carbohydrates. Two infants, breast fed by thyrotoxic mothers, had 10 to 12 loose stools daily. While they were not ill, they failed to gain until weaned.

Parenteral Infections

Parenteral infections in infants are frequently complicated by diarrhea. This is true in all seasons, and is more likely to occur in the first year, and rarely after the second. It may occur with an upper respiratory tract infection, with otitis media, with pyelonephritis and with almost any infection. Diarrhea sometimes starts before the infection has become manifest, or it may not begin until several days after the onset of the infection. Diarrhea associated with infection is less frequent if the infection is rapidly controlled with antibiotics, and improves more rapidly if the infection is quickly controlled.

The explanation for the occurrence of diarrhea with parenteral infection is not clear. The theory formerly held was that in the presence of fever or infection, and during severe summer heat,[34] there occurred a diminished secretion of enzymes into the gastrointestinal tract, resulting in incomplete digestion and poorer absorption of foodstuffs; also, that an alteration of the bacterial flora of the intestinal tract, with ascent of enteric saprophytes, occurred.[17] These bacteria were supposed to liberate toxins which caused the diarrhea. It was also believed[34] that the liver was less efficient during infection in detoxifying or metabolizing proteoses and peptones which were irritating.

Although there is confirmatory evidence of the reduction in enzymes and the migration of enteric bacteria, nevertheless a causal relation between these changes and the etiology of diarrhea is not firmly established. The leveling off[37] of the peak incidence of diarrhea in the summer months, as already mentioned, suggests that the explanation for diarrhea during parenteral infection and during the hot summer months is that there is a greater likelihood of the infant to succumb to infection, and an increased chance of introducing infection with inadequately prepared food.

Emotional Disturbances

It is difficult to ascribe the etiology of diarrhea to emotional disturbances in infancy. Nevertheless, Prugh and Schwachman[42] recently indicated that the diarrhea associated with a syndrome commonly considered to be celiac disease was associated with emotional disturbances in the family. In the older child there is ample evidence that diarrhea can result from an emotional upset. The child with ulcerative colitis undoubtedly varies the frequency of bowel movements with her emotional status and may respond favorably to psychotherapy. One such patient volunteered that she has to have the bed pan the moment her mother arrives. When asked whether it was equally true when her father came, she volunteered: "No, you see he can't get the bed pan fast enough." Many children have diarrhea before important examinations, or in anticipation of their first party or first date.

Irritation of the nervous system with subsequent gastric ulceration was described by Cushing.[11] I observed a four year old boy who had a bleeding duodenal ulcer on the fourth day after removal of a brain tumor. Diarrhea due to shock has been well documented, and gastrointestinal lesions have been reproduced experimentally by Penner and Bernheim.[12] The frequency of gastric erosion and ulceration in bulbar poliomyelitis is relevant.

Summary

Summarizing, the etiology of diarrhea is primarily associated with infection, enteral or parenteral; with alimentary causes, such as overfeeding, improper feeding, sudden changes in diet, introduction of new foods; with constitutional factors: allergy, celiac syndrome, cystic fibrosis of the pancreas; and with neurogenic and emotional disturbances.

Tables 3, 4 and 5, slightly modified by the author from Cheever,[5] summarize the etiology of diarrhea, and compare the effect of preformed toxins with specific bacterial infection.

PATHOLOGY

Shigellosis, salmonellosis, amebiasis and, to some degree, typhoid produce an acute inflammatory process of the intestinal mucosa with excessive mucus secretion, purulent exudate, edema, ulceration and hemorrhage. The various strains of *Escherichia coli* produce slight congestion of the mucosa and hypertrophy of Peyer's patches. The pathology in this condition is minimal, considering the fulminating course of the disease in the newborn and in the very young. In noninfectious diarrhea there is little correlation between the severity of the illness and the findings in the gastrointestinal tract. Pathologic changes else-

TABLE 3. *Bacterial Agents Causing Acute Diarrheal Disease in Man*

ORGANISM	DISEASE	INCUBATION PERIOD
Vibrio comma	Cholera (none in U.S.A.)	1–3 days
Shigella	Bacillary dysentery	1–3 days
Salmonella	Gastroenteritis	12–24 hours
Staph. aureus	Gastroenteritis	2–4 hours
Strep. faecalis	Gastroenteritis	2–18 hours
Escherichia coli (0–55, 0–111, etc.)	Gastroenteritis	2–4 days, up to 14 days

TABLE 4. *Comparison of Intoxication and Infection*

CATEGORY	INTOXICATION	INFECTION
Pathogenesis	Ingestion of preformed toxin	Multiplication of organism within gut
Incubation period	2–6 hours	6–72 hours
Duration of symptoms	Hours	Days
Vomiting	Severe	Usually not marked
Convalescence	Rapid	More prolonged
Communicability	Noninfectious	Infectious

TABLE 5. *Salmonella Types Most Frequently Isolated from Cases of Gastroenteritis in Man (U.S.A.)*

	ANTIGENIC GROUP	CHIEF ANIMAL RESERVOIR
S. typhimurium	B	Rodents
S. newport	C2	Poultry
S. orenienburg	C1	Poultry
S. choleraesuis	C1	Swine
S. anatum	E|	Poultry
S. montevideo	C1	Poultry
S. panama	D	Undetermined

where are uncommon. Thrombosis of a cerebral sinus or a renal vein, and, in the very young, occasionally calcification of the renal epithelium are noted. Fatty infiltration in the liver is common.

The most severe effect of diarrhea is a disturbance in the physiopathology, especially as it concerns the water and electrolyte balance. When diarrhea is very mild, there is little or no metabolic disturbance. The loss of undigested food, fluids, and electrolytes from the gastrointestinal tract is insufficient to produce clinical disturbance. If the diarrhea becomes severe and continues, a greater loss of food substance and electrolytes and water results in hemoconcentration, acidosis and, if uninterrupted, in peripheral shock and circulatory collapse. In this stage there is really a state of metabolic bankruptcy.

Metabolic Alterations

In mild or moderate diarrhea most of the protein is split and absorbed. In the more severe diarrhea there is usually an increased nitro-

gen content of the stool. Fat digestion may be disturbed. Instead of the usual excretion of 5 to 15 per cent of the ingested fat, as much as 25 to 75 per cent may be found in the stool in the form of neutral fats, fatty acids, and soaps. The carbohydrates are generally absorbed unless there be large particles of starch-containing foods which may be excreted. These digestive alterations are due in great measure to the insufficient time exposure to enzyme activity, because of the shortened period that food remains in the gastrointestinal tract and, to some degree, because of diminished enzyme secretion. Instead, peristalsis was so increased in one infant that ingested charcoal appeared in the stool within four hours. Further metabolic disturbance is evidenced by fatty infiltration into the liver and reduced glycogen. Altered assimilation of glucose is suggested by the finding of severe hypoglycemia in some, and hyperglycemia in other severely dehydrated infants. Both flat and diabetic type blood sugar curves have been observed, and glucose injected intravenously may be excreted in excess, whereas, after recovery, the same amount of glucose injected at the same rate is metabolized. The excessive excretion of glucose in this condition may be associated with defective tubular reabsorption.

Accumulation of organic acids has been observed. Lactic acid acidosis has been reported by Clausen.[9] Ketone body excretion in the urine is uncommon.

While absorption of food substances is disturbed during diarrhea, Chung,[6, 7, 8] Holt et al. have demonstrated that with an increased intake of food substances such as fat, there is also a greater retention.

Perhaps most important for the welfare of the infant is the water and electrolyte loss. This occurs largely through the gastrointestinal tract. Three hundred to 500 cc. of water may be expelled with stool in a day, which is five to 10 times normal. With this water there is an excessive loss of electrolytes, preponderantly sodium, in the stool and chloride with vomitus. While sweat is hypotonic, it may nevertheless contribute to the loss of both water and electrolytes, especially during fever or when the external temperature is high.[15] Water loss through sweat and insensible perspiration may account for the rapid loss of weight and dehydration in some infants with relatively few stools.[15] Darrow[14] and his co-workers have called attention to the disturbance of cellular physiology which results in a replacement of intracellular potassium by sodium. Darrow regards the acidosis in these children to be due largely to this loss of sodium from the extracellular to the intracellular compartment. Little is known of the role played by skin and bones in this condition,[2] but in all probability it is not a minor one in electrolyte balance. Alkalosis is a rare finding in diarrhea, but has been reported by both Gamble[21] and Darrow[13] in infants with

congenital defect of chloride absorption from the bowel resulting in excessive loss of fixed acid.

TREATMENT

If the child has an increase in stools from the usual one or two to three or four daily, and if the child is alert, playful and willing and eager to take food, improvement usually follows without special treatment, or merely with a reduction in the total amount of food; or by dilution of the formula with water; or by substituting low fat milk with 2 to 3 instead of 5 per cent added carbohydrate. If the child's diet contains fruit, vegetables and cereals, the fruit and vegetables might be excluded. On such a regimen improvement follows in a day or two, and return to the previous formula or foods is made progressively within the next few days. If this therapy is unsuccessful, or if the diarrhea becomes worse or is worse from the beginning, a period of six to 12 hours of starvation is suggested, after which a small amount (1 to 2 ounces) of a low fat or skimmed milk formula is offered every two hours, and an increase made every 12 to 24 hours, permitting three to five days for re-establishment of a full diet. The mild and moderate diarrheas are those which seem to respond to the various commercial pharmaceutical preparations recommended for the treatment of acute diarrhea in infants and children.

In all cases of diarrhea attention should be given to possible infection, enteral or parenteral, and suitable therapy instituted if such is present. Cure of the infection is often accompanied by correction of the diarrhea without dietotherapy, although a combination of dietotherapy and antibiotic therapy is indicated in most instances of diarrhea accompanied or caused by infection.

In treating an infant with mild diarrhea, if improvement is not rapid, the child should be observed carefully for constitutional symptoms. Unless controlled, the diarrhea may progress to the same point that the severe diarrhea does, in a variable period of time from 12 hours to two or three days, when the infant becomes dull, disinterested, irritable or drowsy. The child may become febrile, the eyes lose their luster, the skin turgor become poor, and the abdomen distended. Convulsions and coma may ensue. The weight loss due to diarrhea, insensible perspiration and starvation may amount to a pound or more in a day. The child is dehydrated, acidotic and in shock, or in an impending state of circulatory collapse. Such a child is in urgent need of treatment for shock, and will not survive unless the circulation is rapidly restored. All other therapy in cases of this type is secondary and is dependent on the successful correction of the state of shock and circulatory collapse.

There have been many outlines for treatment of this disease. The first comprehensive plan was devised by Park[38] and Powers.[41] This was followed by the introduction of continuous intravenous therapy[29] and later by the introduction of antibiotics and vitamins. Throughout this time there has been much discussion of the type of electrolyte indicated. Most of these recommendations were based on the studies of Gamble,[19, 20] Butler,[4] Hartman[26, 27] and Darrow[16] and their co-workers.

The treatment for intestinal intoxication begun in 1929 and presented in 1931[29] has been used with slight modifications at the Mt. Sinai Hospital, New York City, to date. The regimen as modified by Drs. Horace L. Hodes and Samuel Karelitz follows.

Intestinal Intoxication

The treatment of patients with gastrointestinal disturbances severe enough to require hospitalization is fairly uniform in most clinics. Multiple factors involved in therapy indicate that until study modified by experience has produced in the individual physician capacity to manage these patients well, a guide to the effective treatment of them is important.

The following suggestions constitute a regimen which at Mt. Sinai Hospital has produced a low morbidity and mortality. This regimen constitutes the basic plan of therapy of intestinal intoxication on the Pediatric Service. It may be modified as required by unavoidable circumstances or by sound judgment of the individual case, preferably after consultation with an attending pediatrician.

The chemical determinations requested may be postponed if obtaining the necessary blood will cause a dangerous delay in beginning treatment or if the patient's condition is too critical for the handling necessary to obtain the specimens.

A. ON ADMISSION

1. *Physical Examination:* Observe and record circulatory status, including blood pressure and cardiac findings, skin turgor, weight, temperature, and presence of infection. Obtain electrocardiogram when indicated.
2. *Blood Determinations:* Carbon dioxide content, chloride, hemoglobin, hematocrit and blood grouping in all infants. When possible, also a complete blood cell count, sodium potassium, total protein, blood urea nitrogen and sugar.
3. *Stool Examination:* Examine smear of stool for presence of white and red blood cells. Plant stool cultures as per routine method. Leave large specimen of stool in ice box in bacteriology laboratory for possible isolation and typing of *E. coli.*

B. TREATMENT FIRST DAY

1. Give penicillin. When a specific infection is discovered, an appropriate antibiotic is administered.
2. Nothing by mouth for 12 hours.
3. Begin continuous intravenous drip, using Ringer's lactate solution with 5 per cent glucose. Administer 20 cc. per kilogram the first half-hour and 15 cc. per

kilogram the second half-hour. After the first 35 cc. of Ringer's lactate solution and 5 per cent glucose have been given, substitute half-strength Ringer with 5 per cent glucose and adjust intravenous drip to deliver a total of approximately 150 cc. per kilogram of body weight per day. This proportion may be varied if indicated by chemical findings.

4. If the child shows evidence of poor circulation, plasma or blood should be given. If the hemoglobin concentration is less than 11 gm., give a transfusion by drip of 10 to 20 ml. of blood per kilogram of body weight. Otherwise, plasma may be given in the same way, using a dosage of 15 to 30 ml. per kilogram.
5. When carbon dioxide is known, calculate the amount of sixth-molar sodium lactate necessary.
   2 × weight in kilograms × (45 − CO_2 vol. %) − cc. sixth-molar lactate. Thereafter continue intravenous drip with 5 per cent glucose in half-strength Ringer or half-strength saline solutions.
6. After the first 12 hours offer 5 to 10 cc. of water by mouth every 2 to 3 hours unless vomiting has continued.
7. Solutions containing higher concentration of potassium are given at the end of 12 hours of therapy if the child is voiding freely. A solution is made by adding one 20-cc. ampule of 3.7 gm. of potassium chloride to 980 cc. of glucose in water (this solution will contain 50 milliequivalents of potassium per liter). An amount of this solution calculated to give 3.4 milliequivalents per kilogram of body weight per day is then administered orally if possible or by slow hypodermoclysis. This is to be included in the estimation of total quantity of fluid required.

C. SECOND DAY AND THEREAFTER

1. Continue intravenous fluids until approximately two-thirds of daily requirement is taken orally. Approximately 150 cc. per kilogram is required. If the circulation is good, fluids may be given subcutaneously if the intravenous route is not possible.
2. Increase water by mouth to 10 to 20 cc. every 2 to 3 hours after 12 hours or on the second day and daily until the fluid requirement is satisfied.
3. At the end of 36 hours, if the general condition is good, diarrhea improved, no abdominal distention, and the child is hungry, start formula, giving 10 calories per kilogram divided into 12 (every 2 hours) feedings.
   Formula: 1/3 water, 2/3 milk, 5% sucrose — 20 cal./30 cc.

D. DAILY INCREASE

Increase formula by 10 calories per kilogram per 24 hours until 100 calories per kilogram is reached. Increase water by 5 to 10 cc. given with the formula unless the appetite is small, when water should be given after the formula. Reduce feedings to 8 and later to 6 as indicated by the appetite. Discontinue parenteral fluid when oral intake satisfies the daily fluid requirement.

E. TRANSFUSIONS: ABDOMINAL DISTENTION, HYPOTHERMIA

Repeat the plasma or blood transfusions as indicated. Abdominal distention and hypothermia mean that food should be decreased. Distention means that oral feedings should be corrected, reduced or eliminated. During treatment, repeat physical examination and blood chemistry studies, hematocrit and hemoglobin, as indicated.

F. VITAMINS

Multiple vitamins, especially B and C, are added to intravenous therapy and supplied by mouth after parenteral therapy has been discontinued.

NORMAL SERUM VALUES (milliequivalents/liter)

Na	142	Cl	103	
K	5	HCO_3	27	Gm. protein × 2.43 — mEq./liter
Ca	5	Prot.	16	Co_2 vol.%/2.2 — mEq./liter
Mg	3	Acids	9	

REPAIR SOLUTIONS

Ringer's	*Gm./100 cc.*	*mEq./liter*		*mEq./100 cc.*
NaCl	0.86	Na	148	14.8
KCl	0.03	K	4	0.4
$CaCl_2$	0.033	Ca	6	0.6
		Cl	158	15.8
Ringer's Lactate				
Na lactate	0.31	Na	131	13.1
NaCl	0.60	K	4	0.4
KCl	0.03	Ca	4	0.4
CaCl	0.02	Cl	111	11.1
		HCO_3	28	2.8
Molar/6 Sod. Lactate				
Na lactate	1.868	Na	166	16.6
		HCO_3	166	16.6
Darrow's Solution				
Na lactate	0.59	Na	122	12.2
NaCl	0.40	K	35	3.5
KCl	0.26	Cl	104	10.4
		HCO_3	53	5.3

Whole Cow's Milk (approximate milliequivalents/liter)

Na	24	Cl	26
K	36	P	28
Ca	73		

In the outline of therapy here described, as in the regimen recommended by Park,[38] Powers,[41] Darrow,[20] Poleri,[40] the emphasis is on the need to replenish the water and electrolyte deficits by the intravenous route. Thereby the circulatory volume is restored, the blood concentration is corrected, renal function is stimulated, and the circulatory collapse and shock are eliminated.

Although some infants have hypoelectrolytemia, others have hyperelectrolytemia; while some are more acidotic, others are less so; although the degree of dehydration and renal function varies, some are oliguric, others anuric—the choice of an electrolytic solution to be injected at first is not as important at the beginning of therapy as it is later. We have found the Ringer's lactate solution with 5 per cent added glucose quite suitable until the blood chemical findings are known, after which a more accurate attempt to correct the acidosis can be made. The formula suggested for determining the amount of sixth-molar lactate is by no means precise, and we attempt to raise the carbon dioxide content to 45 volumes per cent, since rapid overcorrection may lead to tetany. We have observed this phenomenon clinically only once in the past 25 years. Possibly routine blood calcium determinations would reveal more frequent occurrence of chemical tetany. The reduction in electrolyte content of the fluid injected after the first day has eliminated manifest edema, although excessive expansion of

the extracellular space was demonstrated by Katcher et al.[31] We have not found this to be harmful.

Potassium had not been given regularly; nevertheless the mortality had dropped to below 5 per cent. Since the administration of potassium is physiologically correct, it is added after adequate urinary flow has become established. Blood or plasma is given to help maintain a better circulation. Though neither is needed in all instances, infants do better if given blood or plasma. Other plasma expanders have not been used with regularity.

Feeding

The introduction of milk is based on our observations that the amount given is more important than the quality. Some prefer one or another type of milk, but it has been our experience that, except when dealing with an allergic or celiac child, the type of formula does not seem to be very significant. The increase of food is made slower than is recommended by some, but this is done deliberately to avoid the occasional recurrence of the entire alimentary intoxication syndrome ascribed to too rapid increase of milk in the very young infant. Older children and those who progress well are more likely to receive daily increases of 20 calories per kilogram.

Antibiotics

The choice of antibiotic must depend on the type of infection present and on those currently prevalent in the area of the sick child's home. In some parts of the world preference is given to antibiotics effective against gram-negative bacilli, while in our hospital we have found it advisable to start routinely with penicillin and add the antibiotic best suited for the type of infection discovered.

Responsibility for Direction of Treatment

Even the person who has had a long experience is unable to judge the severity of the illness of every child with diarrhea. The rate of recovery may not depend on the degree of deviation of the chemical data from the normal. The infant who looks very ill and/or has severe electrolyte disturbance and dehydration may respond more rapidly than the one who appears to be less sick and has less blood concentration and acidosis. For these reasons the attending and resident staff are more likely to be successful in a higher percentage of infants if they follow a regimen of therapy, at least until the shock, blood concentra-

tion, acidosis and renal function are corrected. The finer correction of the electrolyte imbalance may be safely left to the kidney. The error of overestimation of severity of the child's illness is far less serious than underestimating urgency of the need for intravenous therapy.

Treatment with Limited Laboratory Facilities

The treatment of severe diarrhea with dehydration must be simple enough so that, when necessary, it can be administered with limited laboratory facilities or without the help of a chemical and bacteriologic laboratory. Admittedly, this is not the ideal situation for successful management of this condition, but it is possible to attain excellent results if intravenous therapy is begun early, blood or plasma is supplied, and suitable antibiotics are given. If the red blood cell count, the hemoglobin and the hematocrit are determined before treatment is started, and repeated later, one can determine whether dehydration has been corrected and whether this correction persists. It may be said categorically, if the infant's color is good, the skin is moist, the tissue turgor good, the blood pressure normal, and the hematocrit, hemoglobin and red blood cells in proper relationship, and if the urinary output is adequate, that treatment is progressing favorably and that recovery is almost certain.

Diarrhea in the Older Child

In the older child diarrhea is usually associated with specific infection or with a dietary indiscretion. For the latter a temporary period of reduced food intake or possibly a six to eight hour period of starvation is usually helpful. Antispasmodics, paregoric and other drugs may relieve cramps and tenesmus, and reduce the frequency of bowel movements. Cathartics or purgatives are not indicated.

When the diarrhea begins abruptly and is associated with vomiting, abdominal pain and fever, it is due to an acute infection, or intoxication with salmonellosis, shigellosis, staphylococcus toxin, and rarely with botulism. In such instances the disease is treated symptomatically, while the specific etiologic agent is determined. Antibiotic therapy is introduced as indicated. If the vomiting and diarrhea are so severe that the child becomes dehydrated and collapses, intravenous therapy is urgent and must be continued until the child is no longer toxic, and is able to tolerate fluid by mouth. In the older child the increase in food may be faster than in the infant. Beginning with a fluid diet, it is changed to a soft diet, and to a full diet as rapidly as seems indicated.

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1176 Fifth Avenue
New York 29, New York

ULCERATIVE COLITIS IN CHILDREN

ALBERT S. LYONS, M.D.

The incidence of ulcerative colitis in children is higher than is generally realized. In 1941 Elitzak and Wideman[5] studied 23 children with ulcerative colitis. Jackman, Bargen and Helmholz[9] reported that approximately 11 per cent of 871 patients with ulcerative colitis were children one to five years old. Willard, Pessel, Hundley and Bockus[21] found that 24.2 per cent of their patients were between the ages of 10 and 19. Over an 18 year period Kirsner, Raskin and Palmer[12] were able to study 80 children with ulcerative colitis. Usually the disease affects but one member of a family. Familial incidence, though reported sporadically, is apparently not particularly noteworthy.

PATHOLOGIC FEATURES

Ulcerative colitis may be divided according to its gross distribution throughout the colon into three forms. Universal colitis, in which the entire colon from cecum to anus is involved, is the most common. When the disease is limited to the rectum and left colon, sparing the cecum, ascending and transverse colon, the term "left-sided colitis" is used. In right-sided colitis, comprising approximately 5 per cent of all cases, the rectum and sigmoid are spared and parts or all of the descending, transverse and right colon are affected.

The disease may start in any segment of the colon, but is commonly believed to start most often in the rectum and sigmoid. It may be that most reports implicate the rectum as the region of onset because this area is more accessible to digital and visual examination. Once begun, the process may proceed proximally or distally. The pathologic

From the Surgical Service of the Mount Sinai Hospital, New York City.

course apparently is the same in children as in adults, beginning in the mucosa, but rapidly involving all coats of the colon in the inflammatory process, advancing to ulceration, penetration, perforation, abscess, fistulization, fibrosis, "polyposis" and even malignant change. The terminal ileum may show associated disease, called "backwash ileitis." Occasionally the small bowel enteritis may be more extensive and may resemble regional enteritis. This association is not any better understood in children than in adults.

It was formerly assumed that advanced changes (e.g., fibrosis, pseudopolyposis) were irreversible, but isolated reports indicate that reversion to normal-looking bowel, although uncommon, can occur. This is particularly to be borne in mind with children. It is also well to emphasize that pathologic changes in the colon may not parallel the clinical course.

DIAGNOSIS

Onset and Course

The clinical syndrome of ulcerative colitis is virtually the same in children as in adults. The onset may be acute and fulminating with severe toxemia, watery and bloody diarrhea, high fever, and rapid progression to a moribund state or to one of the severe complications of the disease. In altogether different manner, the disease may be so mild in its beginnings and so insidious in its advance that marked changes in the bowel may develop before one is aware that the intermittent mild diarrhea represents ulcerative colitis. Diarrhea may be absent altogether, or constipation may be noted. On the other hand, the most common type of onset is between these two extremes, with a subacute diarrhea, often bloody, persisting longer than anticipated and associated with varying degrees of abdominal discomfort, malnutrition, fever and anemia. The onset may sometimes be preceded by an acute upper respiratory infection, a specific intestinal infection or severe emotional trauma.

No matter what the manner of onset, the disease in the majority of instances runs a course which, though disabling for periods of time, permits the development of the child. However, when the disease continues in activity in the prepubertal and pubertal years, marked retardation in physical development is a real possibility. Examples of infantilism as a result of ulcerative colitis have been reported, and there are even instances of severely underdeveloped young adults whose earlier ulcerative colitis in puberty had gone unrecognized clinically.

The course may be one of remissions and recrudescences through the years; it may persist steadily in chronic low grade fashion; it may become arrested and the patient never have a recurrence. It is difficult

to know what proportion of cases falls into each of these categories, but together they probably include the great majority of patients. There are, however, a definite number of children whose illness, resisting all measures and advancing into intractable, disabling complications, leads either to death or to permanent invalidism.

It is also of particular importance to realize that the severity of pathologic change in the colon may bear no direct relation to the seriousness of the clinical course. Some of the sickest, most gravely ill children with prostrating toxemia may have minimal mucosal inflammation and ulceration, and, contrarily, children with advanced "pipe stem" colons may look relatively well and well nourished.

Diagnostic Methods

Endoscopy is essential to determining the diagnosis. Whether one uses general anesthesia in order to pass the instrument in children depends on one's own preferences, on the cooperation of the child and on the amount of disease affecting the anus. Individual exigencies should determine the choice and the least upsetting method should be used. In some children this means general anesthesia without trying to do without it, whereas in others the better method is simple passage of the endoscope after application of a topical anesthetic, in an alert, cooperative patient.

In any case, no more than the rectum need be visualized, and usually the first few inches in children will yield the important information. The bowel wall may be friable and therefore in danger of injury or perforation. Gentleness is therefore essential. If one has available the proctoscopes of smaller caliber, they are often more comfortable to the unanesthetized child, but even the regular adult sizes may sometimes be introduced easily. One should be prepared to take smears and aspirations from the lesions seen, for sometimes only by this direct means can amebas be found.

The state of the mucosa in the early stages may merely show hyperemia with friability, yielding blood on wiping with cotton. Small punctate hemorrhages or tiny superficial abscesses may also be noted. In the more advanced states there may be ragged ulcerations of varying sizes covered by white or gray exudate with hypertrophy and redness of intervening mucosa. Characteristically, in ulcerative colitis, there is diffuse involvement of the lining so that there is no normal-looking mucosa between lesions. Sometimes the entire lining has been cast off except for a few intermediate islands of mucosa, and one gets the erroneous impression that the small islands are polyps set in intact mucosa. Blood, pus and mucus may fill the endoscope and require aspiration or wiping in order to allow visualization. Long-standing

disease may be associated with polypoid fronds projecting into the lumen. These usually represent hypertrophied mucosal tabs often bounded by areas in which the mucosa has been denuded. These polyps are not true adenomas and are usually referred to as pseudopolyps. True adenomatous polyps, however, can also be found, and it may be extremely difficult or impossible to determine the nature of the polyps seen. This sometimes causes difficulty in differentiating familial diffuse polyposis from quiescent but long-standing ulcerative colitis associated with many polyps.

In "right-sided" colitis of course the rectum is normal. Under these circumstances endoscopic examination is of prime importance, for it is chiefly upon these findings that the decision depends as to the feasibility of making an anastomosis of ileum to rectum. Here it *is* important to try to determine how far up the mucosa is normal.

Roentgen examination of the colon by barium enema may reveal normal findings, except for spasm, in the early stages when pathologic changes in the mucosa are relatively superficial. Definite disease may be present with normal roentgen findings, and abnormal findings, no matter how minor, are indications that the disease is well established. For example, once loss of haustrations is seen, the process is already advanced. Other findings which appear as the disease advances are feathering of the mucosa, loss of distensibility, luminal narrowing, and polyposis. In ulcerative colitis demonstrated on barium enema it is wise to obtain small bowel studies to search for regional enteritis, for the presence of small bowel inflammation has bearing on prognosis, treatment and follow-up evaluation.

The principal conditions which must be differentiated from ulcerative colitis are amebic dysentery, bacillary dysentery, paratyphoid fever, tuberculous enteritis, deficiency states and nonspecific gastroenteritis.

MEDICAL TREATMENT

Since there is no known specific therapy for ulcerative colitis, nor any special nonspecific measure which is invariably effective, the treatment of each child must be highly individualized. Each modality of therapy must be examined as to its suitability for the particular child and its parents. At the same time one should be willing to make use of whatever methods and drugs are available in the medical armamentarium, varying the treatment according to the needs. Enthusiasm and caution in equal measure are valuable assets in the clinician who has the multifaceted problem of managing the child sick with ulcerative colitis.

Everything that is done during treatment has psychologic repercussions. It must be realized that with less understanding than an adult

the child is often unable to distinguish between diagnostic and therapeutic procedures. Overburdening him must be avoided, as long as there is no sacrifice of vital investigation or treatment. Comfort and cheerfulness are the goals to be sought and are the keynotes of therapy.

Hospitalization

Hospitalization is usually necessary for the very ill and often yields better results even for those not so seriously afflicted. The mere removal into a different environment may be salutary, but only if the new surroundings are cheerful and comfortable, and include diligent, gentle and motherly nursing care.

General Analeptic and Nutritional Measures

Transfusions are of inestimable value in helping to restore depleted blood hemoglobin and other proteins. Intravenous saccharated iron has been advocated by some with excellent results in combating anemia. Vitamins by mouth had best be of the type without oily taste or smell and should be discontinued quickly if poorly tolerated or resisted, using instead parenteral vitamins in therapeutic doses. Liver extracts are sometimes useful as an adjunctive measure. Water and electrolyte imbalance may try the skill of the clinician in adopting vigorous and yet safe measures. Parenteral fluids in adequate quantities as in any severe diarrhea are usually needed. (These measures are considered in the clinic by Dr. Karelitz in this symposium.)

Diet

It is usually wise to avoid foods known to be irritating to the intestinal tract, giving at the start a bland diet which is as nutritious as possible. Sensitivity to milk or other foods of course may alter the original plan. Small frequent feedings may be better tolerated than larger concentrated amounts. In those with immediate tenesmus after eating, longer periods may be needed. The aim, however attained, is good nutrition, and if the patient can tolerate the foods he should be encouraged to eat what he can. Persistence in the use of low residue regimens may do more harm than good. Whatever will pique a child's appetite should usually be allowed.

Sedative Drugs and Antispasmodics

There is a long list of well known narcotic, analgesic and antispasmodic drugs, as well as numerous new preparations, which have proved

useful in helping to allay pain, diarrhea and tenesmus. None is sufficiently or outstandingly successful to warrant singling out for mention, but each has its special advocates, and it is well to be persistent in trying one after the other if necessary in the search to obtain comfort for the patient. The most commonly effective time to use antispasmodics is before eating, to lessen the reflex urgency, which may be particularly annoying.

Antibiotic and Chemotherapeutic Drugs

There is virtually no agent against bacteria which has not at some time been reported to be exceptionally successful in the treatment of ulcerative colitis. Each brilliant promise has so far gone unfulfilled. Yet one is sometimes so impressed by the spectacular response to one or another of the antibacterial agents that they must of necessity be included in the over-all therapeutic program. Among the sulfonamides which have been useful are sulfaguanidine, salicyloazosulfapyridine, succinylsulfathiazole, phthalylsulfathiazole. The antibiotics include chloramphenicol, tetracyclines, penicillin, streptomycin and others.

These drugs should be administered in combinations according to the response and needs in each patient, chiefly on empiric grounds. When one seems ineffective, another may be tried. Sometimes a localized abscess formation permits culturing and sensitivity studies to help determine which drug is more likely to be effective, but the information so gained is not always paralleled by the response in vivo. One must of course always bear in mind the limitations, dangers and contraindications in the use of each antibacterial agent.

Hormones

The recent use of ACTH, cortisone, hydrocortisone and their analogues in the treatment of ulcerative colitis is difficult to evaluate. Enthusiastic champions with glowing reports as well as violent opponents with extreme derogatory experiences have emerged. My own experience and observation suggest that the use of these drugs has often proved a great boon. They have helped, for example, in restoring the critically ill patient with acute disease to a safer condition which would permit operation; in halting the rapid, inexorable course of an exacerbation in chronic disease; in contributing to the recovery of seemingly intractable colitis by adding a sense of well-being and appetite. But though there are successes, there are also failures. Even if a good response occurs, relapse may ensue when the drug is withdrawn. Furthermore, not only may the poor clinical course be unchanged, but to

this may also be added the disturbances in electrolyte pattern and any of the serious complications of the steroid group (e.g., perforation).

In general, I think it worth while to administer these hormones, particularly to the severely ill children, watching always for complications and stopping if salutary response is absent or inadequate. If any surgical procedure is to be performed, the drug must be continued throughout the operative and postoperative days or else stopped long enough in advance (for approximately two days for each week of therapy) lest adrenal insufficiency and collapse result.

Desoxycorticosterone may sometimes aid in achieving hydration of tissues, particularly in patients who have had severe dehydration and peripheral collapse due to sudden outpourings of gastrointestinal fluids. The usual precautions to avoid hypernatremia and hypokalemia should of course be observed.

Rectal Instillations

Soothing suspensions and solutions are sometimes instilled into the rectum to attempt to lessen tenesmus and anal pain. Kaolin and warm oil are among the substances most commonly used. I have seldom found them helpful and contrarily have sometimes noted aggravation of discomfort and tenesmus. On the other hand, protective powders and ointments on the anal skin may be greatly needed. Occasionally an anesthetic cream applied at the anal verge or introduced into the canal will relieve the severe pain caused by anal inflammation or ulceration. If the patient's condition permits, a warm tub bath may be gratifying in allaying anal sphincter spasm.

Psychotherapy

There are psychotherapeutic implications in the management of all children with ulcerative colitis. The precise role of the psychic factors has not been clearly established, but no matter what their place in pathogenesis, they are highly important in treatment and may even be the determining feature in achieving recovery.

The use of intensive psychotherapy by a psychiatrist in the child with mild, early or quiescent colitis may be of great value, but formal or deep penetration is not usually suitable when the child is seriously ill. However, there is no reason to miss following up a trail which the child may inadvertently open. Also, even resolution of a superficial conflict can lead to great improvement in the patient's emotional state.

Although aid should be enlisted whenever needed, the most important part in the psychologic management usually falls to the pediatrician. Included with his persistence in attempting therapeutic methods

should be alertness in seeking clues to possible psychic difficulties. Furthermore, by patience in handling the entire family constellation, by preserving balance in order to avoid dogmatic rigidity, and by gentle firmness in maintaining control of the entire regimen, the clinician may sometimes achieve psychotherapeutic results beyond his expectations.

SURGICAL TREATMENT

Indications for Operation

Wide differences of opinion exist on the frequency of the need for operation in adults with ulcerative colitis. The frequency varies from 30 to 10 per cent. Concerning children, there is almost universal reluctance to submit the patient to a definitive procedure which involves performing an ileostomy. This position is understandable and even wise, provided it does not stem from ignorance and mistaken ideas on the nature and details of management of ileostomy. On the other side, ileostomy should not be embraced as a simple mechanical contrivance for the cure of most ulcerative colitis. Operative therapy should not be used if the patient can manage to carry on for long intervals and continue to develop, nor too long delayed by prejudice in the face of advancing or debilitating disease.

More important than the state of the bowel is the condition of the patient. *The two primary indications for operation therefore are danger to life and invalidism.* The states which usually lead to the necessity for surgery are as follows:

Acute Disease (Danger to Life). ACUTE FULMINATING TOXEMIA. Surgical intervention here should be done before the patient has reached a hopeless state wherein death is imminent.

HEMORRHAGE. Most severe bleeding terminates spontaneously, but unresponding hemorrhage or repeated catastrophic bleeding is an urgent indication for operation.

PERFORATION. Early operation here gives the best chance of survival. Operation sooner or later becomes necessary in virtually all patients who have had a perforation.

IMPENDING PERFORATION. This requires experience and judgment to determine, but once established, operation is urgently needed. The appearance of pain and local tenderness together with a change in the patient's condition is usually indicative of impending free perforation.

Chronic Disease and Complications (Invalidism). INTRACTABILITY TO MEDICAL THERAPY. The child with retarded development, disabling arthritis, resistant skin lesions, persistent anemia or unremitting or repeated disability requires operation if invalided by these or other complications.

STRICTURE. If symptoms of obstruction are present, operation is necessary.

PERICOLONIC ABSCESS. Since this results from slow perforation, definitive surgery is usually eventually needed.

FISTULAS. Internal communications of the colon with other organs or fistulas to the abdominal wall usually require colectomy. Perianal fistulas are indications for colectomy and ileostomy only when extensive and disabling, or when associated with incontinence. When relatively mild, they are best left alone until the acute colitis has subsided. The danger of interference with sphincteric continence by surgical excision is real. Rectovaginal fistula, if extensive and persistent, usually requires ileostomy eventually. Perianal or perineal abscesses should be opened early and widely. They often end as fistulas.

POLYPOSIS. Pseudopolypsis is not necessarily an indication for colectomy, but the association of ulcerative colitis and adenomas of the colon is a dangerous precursor to carcinoma.

MALIGNANCY. When malignancy is detected or suspected in the course of ulcerative colitis, colectomy is urgently indicated. Statistically, the danger of carcinoma of the rectum and colon in children with ulcerative colitis is much higher than in the normal population.

Choice of Operation

The definitive operations available for universal colitis are ileostomy, subtotal colectomy and abdominoperineal resection. The tendency in recent years has been toward performing all three procedures in one stage routinely. Others do the combined ileostomy and subtotal colectomy at the same time, but perform the rectal removal at another sitting. A third, small but steadfast group continues to advocate ileostomy as a single procedure, followed by resection of the colon and rectum at subsequent stages. I believe that each of these methods has its distinct advantages and that one should choose the procedure best suited to each patient.

The philosophy of most surgeons who have experience with ulcerative colitis is that ileostomy is merely a necessary step in the removal of the diseased colon and rectum. Leaving the colon for prolonged periods of time has been fraught with the dangers of active recrudescences and serious complications. On the other hand, leaving the rectum intact in many patients usually seems to have proved safe, provided regular and frequent observation is made. Five adults in our series have even been able to have a successful secondary anastomosis of ileum to rectum with removal of the ileostomy (in later years after ileostomy and subtotal colectomy, when the rectum had returned to a

normal state). Of course this is not to be embraced as a common procedure and is suitable only for selected patients.

I believe that all children in whom operation is required should have at the least a subtotal colectomy (that is, ileostomy and removal of the colon up to the mid or distal sigmoid). For those in poor condition, sometimes the ileostomy alone as a first stage is wise. But the colectomy should follow in the weeks or months ahead when the patient's general restitution has been sufficient. The purpose of the operation is to get rid of the hopelessly diseased organ. Leaving permanently the entire affected colon has proved many times to be too great a risk, and closing the ileostomy with the previously diseased colon still in place has usually been disastrous.

In children in whom the rectum has also shown advanced disease and who are physically and psychically able to withstand the procedure, abdominoperineal resection of the rectum should be performed either at the same time as the colectomy and ileostomy, or at a subsequent stage.

For those in whom the rectum has shown minimal or mild disease, or in whom the advanced proctitis gives evidence of definite regression, abdominoperineal resection should be deferred and the rectum observed over a period of time. Persistent or advancing disease requires early removal, whereas quiescent or healing state would suggest leaving the rectum longer for continued observation. If some day the rectal stump returns to complete normality as indicated by the absence of discharge, and by endoscopy, roentgen study, and later direct examination at operation, then anastomosis of the ileum to rectum can be contemplated. There is some risk involved in thus leaving the previously involved rectum, but less, I believe, than in leaving the entire colon.

The use of the anal canal for anastomosis with the ileum after removal of the entire colon and rectum, as described by Ravitch,[18] has not been embraced with enthusiasm and has been abandoned by the originator for patients with ulcerative colitis. However, in children I think that this operation should be re-evaluated.

Patients with "right-sided colitis" (with the rectum and sigmoid spared) may have an ileoproctostomy and subtotal colectomy in one or two stages. However, for this procedure to be indicated the rectum and sigmoid must be completely normal on endoscopy, roentgen study, and observation at operation. Unless this is rigidly adhered to, disastrous results are apt to occur, such as recurrence, perforation, and death.

Left-sided colitis, in which the cecum and ascending colon, and sometimes transverse colon, are free of disease, should be managed like universal colitis—requiring ileostomy, colectomy and resection of rectum. I have found that transverse colostomy for this disease some-

times presents insurmountable problems, among them difficulty in bag application and odor control, and I strongly advise against its use. With all its disadvantages, ileostomy is to be preferred to a running colostomy.

Preoperative and Postoperative Care

Before ileostomy is performed, the older child should be given some idea of what is to be expected. The fact that he is going to be made well is accented. The need for an appliance is explained simply, but the details are often best deferred. If another child or even a personable young adult with ileostomy visits the patient before as well as after operation, spectacular reassurance may be thereby achieved.

Planning carefully the placement of the ileostomy, even plotting it out on the abdomen or applying a trial bag, is of enormous value later in enabling the proper choice of appliance and in obtaining correct fit. Immediately after operation, either while still in the operating room or shortly afterward before the ileostomy has begun to function, a temporary bag is cemented to the skin. Later, while still in the hospital, the child is fitted with a more substantial appliance which, if possible, he can manipulate himself.

Usually in the early postoperative days, maintenance of water and electrolyte balance becomes the chief preoccupation, whereas the later days seem concerned chiefly with adequate skin protection and appliance management. It is a mistake to think that once the patient goes home the problem has ended. Gross[7] says that "the surgeon who makes an ileostomy assumes a long-term and tedious responsibility in follow-up and care." Particularly with children must the surgeon continue to serve as supporter, guide and teacher for the entire family.

QT Alumni, an organization of patients with ileostomy, first established in New York and later in other cities—notably Boston, Philadelphia and Los Angeles—has become an immeasurable aid to patients, nurses and doctors who deal with the problems of ileostomy. Regular attendance by a child with ileostomy and parents is a most effective means of helping in rehabilitation. One of the especially spectacular results of the ileostomy clubs has been the demonstration to physicians that ileostomy is not a horrible state, but instead a means of restoring the hopelessly ill to useful, cheerful lives which include virtually every normal social, sexual and economic activity.

PROGNOSIS

The outlook of children with ulcerative colitis was generally considered poor, in the past. Elitzak and Widerman[5] in 1941 reported

eight deaths in 23 cases, with seven unimproved or worse. Similarly Jackman, Bargen and Helmholz[9] in 1940 found that over 50 per cent became worse or died in their series of 95 children. Other sporadic reports have added to this impression.

However, these estimates may have to be revised as experience has accumulated. For example, in the recent report by Bargen and Kennedy,[1] approximately 85 per cent of the 139 children had recovered with good follow-up results. Further, Kirsner, Raskin and Palmer[12] state that the mortality rates by all methods of therapy have declined to 5 per cent.

The mortality after operation has also improved spectacularly. From postoperative deaths of 30 per cent and more, the present over-all expectation is 5 to 10 per cent. This may be higher in children who have been permitted to become hopelessly marasmic.

In general, restitution by means of operation has been brilliantly successful, reclaiming the dying and transforming the hopelessly invalided into active, healthy, happy children who achieve full and satisfactory adulthood. Yet it must be realized that the mere presence of an ileostomy is not the only price paid, for complications after ileostomy are not uncommon and in most experience from 20 to 60 per cent of patients of all ages with ileostomy require some form of operative correction subsequently. This should not dissuade the physician from recommending surgery to salvage the dangerously or hopelessly ill child, nor should it lead to performance of ileostomy for less than advanced disease.

Several factors affecting prognosis may be considered.

Onset and Course

Apparently the acutely febrile, toxic patients have the worst outlook. Kiefer[10] reported that 63.6 per cent of patients with acute fulminating disease had poor results, whereas only 22 per cent of those with nonacute forms had unsatisfactory outcome. In most surgical series the greater the proportion of those with acute toxemia, the higher the postoperative mortality.

Extent of Involvement

Some have felt that when the disease is confined to the rectum and sigmoid, the prognosis is better. According to Hern,[8] complete recovery is more likely as long as the ascending colon is free. However, in my own experience the distribution of the disease in the colon bears little relation to severity or outlook.

Small Bowel Involvement

Involvement of small bowel, however, is of great importance. The short segment of inflammation in the terminal ileum—sometimes called "backwash ileitis"—which is found in about 25 per cent of cases, seems to have little bearing on the ultimate course. However, frank involvement of the small intestine above this area, especially if typical of regional enteritis, may presage a resistance to therapy and an advancing course with poor outlook, with or without operation.

Duration

The longer the child is able to survive the ravages of the disease, the better is his outlook. The greatest mortality is usually in the first year of illness. Willard et al.[21] found the best results in adults with the disease for five years or more. However, long-standing ulcerative colitis carries with it a greater chance of malnutrition, retardation in development, and advanced pathologic change in the bowel, including stricture, polyposis and neoplasm. Nevertheless, except for frank neoplastic change, the prognosis generally is based more on the child's clinical adjustment to the disease than on the gross and microscopic findings in his bowel.

Complications

All complications lessen seriously the chances of recovery without surgical extirpation. Free perforation was formerly considered fatal in virtually every instance. In the past decade more and more survivals have been reported, first with treatment by antibiotics alone and more recently by prompt operation. When feasible, ileostomy and colectomy in one stage can often achieve dramatic recovery in the presence of free perforation. Sometimes simple ileostomy and drainage of a fixed area of perforation in a marasmic child is the better policy.

Loss of blood in the stools is most often relatively slow and low grade, producing anemia but not peripheral collapse, but severe, massive hemorrhage with shock does occur. In the majority of instances the bleeding stops spontaneously, although large transfusions may be necessary to maintain blood volume. However, severe persistent hemorrhage may require surgical removal of the bowel. The problem then is to be able to do a radical resection before the patient's condition has deteriorated. This complication therefore has ominous implications.

Internal and abdominal wall fistulas, although needing operation for cure, usually respond well. Perianal fistulas, if mild and single, do not affect the over-all outlook, but multiple anal fistulas, especially when

associated with partial incontinence, are so disabling that ileostomy eventually is needed.

The skin lesions commonly associated with ulcerative colitis (which can be extensive gangrenous ulcerations) vary with the state of the colitis. Even when long-standing and resistant to all forms of therapy, they all become clean and heal well after extirpation of the diseased colon. The arthritic symptoms in ulcerative colitis also parallel the colonic disease. Sometimes this is so severe that it becomes an indication for operation.

Complications involving other organs—liver, kidney, pancreas—of course alter the outlook, but here too reversibility along with improvement in the pathologic state of the colon may occur.

Infantilism and retardation of growth and physical development are complications of serious import, for if they are allowed to continue through the pubertal years, the child may end by being permanently retarded even if the colitis later heals. The years just before puberty are therefore crucial in the child with ulcerative colitis. If somatic stunting associated with active colitis is noted, then the likelihood is great that it will continue unless the colon heals promptly or is extirpated surgically.

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1089 Madison Avenue
New York City

PROTOZOAN AND METAZOAN PARASITOSES OF THE INTESTINAL TRACT

ERNEST CARROLL FAUST, PH.D.

RODNEY C. JUNG, M.D.

Many parasitic infections of the human intestinal tract are more prevalent in warm than in cool climates, a few are more common in temperate zones, and others are cosmopolitan in their distribution. Children are susceptible to all these infections and are peculiarly liable to exposure whenever the elementary rules of personal and group hygiene are not observed. On the average, most of these infections occur more frequently in underprivileged families and in institutionalized children than in those of the same age groups in the general population of the same areas.

For convenience, parasitic infections may be divided into two main categories, viz., those produced by the Protozoa, or one-celled animals, and those caused by the Metazoa, or many-celled animals. Infections of importance in pediatric clinics in North America are caused by the following:

PROTOZOA

The pathogenic ameba *Entamoeba histolytica,** the ciliate *Balantidium coli** and the flagellate *Giardia lamblia*

METAZOA

Roundworms

*Ascaris lumbricoides, Toxocara canis, Trichocephalus trichiurus,** *Enterobius vermicularis,** *Necator americanus* and *Strongyloides stercoralis**

Tapeworms

Hymenolepis nana, H. diminuta, Dipylidium caninum, Taenia saginata and *Diphyllobothrium latum*

From the Department of Tropical Medicine and Public Health, School of Medicine, Tulane University, New Orleans.

In addition, children from Puerto Rico not infrequently suffer from infection with the blood fluke *Schistosoma mansoni*,* and there are occasional cases of accidental infestation with the maggots of filth flies.

Each of these parasitoses which produce lesions in the sigmoid colon, rectum and anal canal (indicated by an asterisk) will be considered with special reference to its epidemiology, pathogenesis, symptomatology, diagnosis, treatment and prevention.

AMEBIASIS

Epidemiology

Entamoeba histolytica, the only tissue-invading ameba of man, is acquired in its encysted stage as a contamination of fingers, food and water, or by personal contact, whereby the cysts get into the mouth and are swallowed. Although dogs, monkeys, rats and possibly other mammals harbor this ameba, almost without exception the source of human exposure is another person who is passing the cysts in his stools. He may have symptoms attributable to the ameba or he may be an "asymptomatic" carrier.[15]

In the southern United States the incidence of amebiasis is moderately high in the cities and considerably higher in rural areas. Surveys in Virginia, Kentucky, Tennessee, Georgia, Mississippi and metropolitan New Orleans all support this conclusion.[12, 13, 14, 26] In the North, Northwest, Southwest, California and Canada amebiasis is on the average considerably less prevalent, but is widely disseminated.[7, 10, 18, 24, 30, 32, 33, 37, 38, 41, 42, 47, 49] This infection is much more common in children's homes, mental hospitals and prisons than in the population at large.[2, 14, 15, 39, 43] Occasionally *E. histolytica* has been diagnosed in infants.[11, 22, 34] Depending on opportunities for contracting the infection, amebiasis builds up from early childhood year by year to a maximum in early adult life.[15]

After the viable cysts of *E. histolytica* have been swallowed they pass through the stomach, and, on reaching a level of the small intestine where the medium is neutral or slightly alkaline, excystation occurs, releasing four small trophozoites. These are carried in the fecal stream into the cecum, where they have the first opportunity to colonize, provided one or more of the amebas lodge on the surface of the mucosa long enough to secure food, grow and multiply. At times the amebas are carried down to the sigmoid colon or rectum, where they initiate primary infection, or they may be evacuated in the feces without making tissue contact. *Entamoeba histolytica* may, at least temporarily, colonize in the glandular crypts, feeding on secretion of mucus, or it may almost immediately invade the mucosa by lytic action. As long as it remains a lumen parasite it is dependent on suitable bacteria to carry

on its metabolic activities, but once it has entered the tissues this association is no longer necessary.[15]

Pathogenesis

The early uncomplicated amebic ulcers consist of pinpoint sites of invasion, with undamaged intermediate areas. The multiplying colony typically digests its way through the epithelial cells down to the muscularis mucosae, but within the submucosa it develops an enlarged base. Usually the depth of the ulcer at this early stage is free of bacteria. Below the mucosa the advancing destructive process may undermine the surface, with sloughing. Some of the amebas in the deeper portion of the ulcer get into mesenteric venules (or lymphatics) and are carried to extraintestinal sites, where they may lodge and initiate secondary foci of infection. Meanwhile progeny will be squeezed out of the neck of the lesion and may produce secondary colonies at lower levels of the large intestine. Later bacterial invasion complicates the picture of the enlarged ulcers as they transform from the acute to the chronic condition.[15]

The greatest number of initial lesions is found in the cecal area (cecum, appendix, ascending colon), while the sigmoidorectal region becomes increasingly involved as colonization of the amebas takes place at this level. Except in overwhelming or fulminating infection the intermediate levels of the colon are not prominently involved in the pathologic process.

In bacterially uncomplicated amebiasis there is relatively little local host tissue reaction, except that repair with normal mucosal cells may proceed almost as rapidly as lytic destruction of the surface occurs, and, in fact, may at times be responsible for spontaneous eradication of the amebas. In contrast, once bacteria have invaded the amebic ulcers there is marginal infiltration of neutrophilic leukocytes and fibroblasts, producing a completely different histopathologic picture.[15, 16]

Symptomatology

Many students of amebiasis have noted that the symptoms produced in adults range widely from little or none to severe dysentery, and may include vague, low grade discomfort referable to no particular anatomic location.[8, 15] In children, possibly because of their lower threshold of susceptibility to psychoneurosis or inadequate ability of self-expression, the infection is usually either below clinical grade or produces relatively characteristic symptoms. A majority of children infected with *E. histolytica* are essentially asymptomatic, and the infection is detected during routine stool examinations. Other children may complain of abdominal

pain and tenderness, typically located in the right lower quadrant of the abdomen; some may present symptoms of diarrhea, acute or recurrent, and a few may have frank dysentery. In areas with endemic *Trichocephalus* infection, as in southeastern Louisiana, amebiasis will often be superimposed on the helminthic infection, and both may contribute to the production of dysentery.[27]

A tender, enlarged liver is sometimes seen in amebic colitis of children, in spite of the fact that they rarely have amebic abscess of the liver.[34, 35] It is not known whether this variety of hepatitis is a manifestation of damage due to absorption of the toxic products of necrosis from the colonic ulcers or to repeated metastatic invasion of the liver by living amebas which fail to colonize. Whatever the cause may be, the hepatic inflammation subsides with successful treatment of the intestinal infection.

Amebiasis of the colon and rectum does not provoke fever, leukocytosis or signs of toxicity unless there is marked secondary bacterial infection. The stools are not purulent; in the dysenteric patient they consist characteristically of clear or slightly cloudy mucus mixed with blood. Amebiasis does not produce eosinophilia, either locally in the bowel or in the blood stream, nor is it responsible for Charcot-Leyden crystals in the stool.

Diagnosis

Specific diagnosis of intestinal amebiasis is based almost entirely on laboratory examination of the stool. One must occasionally prescribe amebicidal treatment on the basis of clinical findings alone in the absence of reliable laboratory facilities, but, if the symptoms do not disappear as a result of therapy, failure is more likely to be due to misdiagnosis than to refractory amebiasis. Four stool examinations made by an experienced technician will detect almost all cases of amebiasis.[15] The specimens should not be obtained on the same day, but on successive or alternate days. If four specimens are negative, a saline purge may provide a liquid specimen containing trophozoites, but the nonpurged specimens should be obtained first.

Presence in the stool of either cysts or trophozoites recognizable as *E. histolytica* establishes a diagnosis of intestinal amebiasis. However, the structure of either form of the organism may not be typical, so that doubt may exist as to the diagnosis. In such a case additional specimens should be examined and culture attempted. While culture is not as satisfactory as stool examination, it may provide trophozoites and perhaps also cysts which may strengthen the diagnosis. Occasionally amebas will grow in culture when stool examination has been negative, but this is not the rule.

The complement-fixation test for amebiasis is not satisfactory for diagnosing the intestinal infection, although it is considered by some workers to be reliable in extraintestinal amebiasis.[15]

In a majority of cases of amebic colitis the rectum and sigmoid colon are not involved; therefore in these cases sigmoidoscopy cannot demonstrate the lesions. The reverse is true in amebic dysentery, as demonstrated by Browne et al. in 98 of 232 cases of active amebic dysenteric patients.[5]

The most striking feature of the amebic intestine on gross examination is the lack of inflammatory reaction in the mucosa unless the dysentery and secondary infection are pronounced. One usually sees ulcers of pinhead size separated by normal mucosa. Each ulcer is surrounded by a narrow, cloudy, raised zone. It may be covered by a "cap" of white necrotic material, which is easily removed with a cotton swab or by preparatory enemas, revealing a bloody base. As the disease progresses, the ulcers become enlarged and confluent and the undermining of their edges becomes evident. Bleeding is considerable in severe cases.

Treatment

A large number of efficient amebicidal drugs are now available, but there is no single drug of choice. The selection depends on consideration of cost, availability, toxicity, efficacy, duration of the course of treatment, promptness of action and type or character of the disease. Drugs effective in nondysenteric amebiasis will frequently fail in the presence of dysentery. The varying incidence of dysentery in different groups of patients with amebiasis is largely responsible for the conflicting reports on the efficacy of the amebicidal drugs used.

When the dysenteric type of the disease is present, efforts are first directed at relieving symptoms. Nonspecific agents such as kaolin or bismuth provide little or no relief and may interfere with diagnosis if given before stool examination. Replacement of fluids and blood may be indicated.

The promptest relief of dysentery is achieved by specific antiamebic therapy. Rapid checking of dysentery has been observed with emetine, the broad-spectrum tetracycline antibiotics, erythromycin, Fumagillin, carbomycin, bismuth glycolylarsanilate and glaucarubin.[8, 15, 31, 46] Rapidity with which a drug controls the symptoms is no index of its ability to cure the infection.

Emetine, one of the most effective in the relief of symptoms, has a low curative index. Because of its toxicity, its parenteral route of administration and its low cure rate, emetine is not recommended as first choice in amebic dysentery. It is suggested that erythromycin[29] or one of the tetracyclines[31, 46] be tried first. Erythromycin is preferable because

of its concomitant effect on the nonpathogenic amebas. The antibiotic selected is given in the usual therapeutic doses concurrently with a preparation reliably curative in nondysenteric amebiasis such as bismuth glycolylarsanilate (Milibis) or diiodohydroxyquin. Failure of a five-day course of the antibiotic to check the dysentery warrants substitution of emetine for the antibiotic.

Emetine hydrochloride is given in a dose of 1 mg. per kilogram of body weight per day intramuscularly for three to five days. The patient should be kept in bed and the pulse and blood pressure taken frequently. Drop in blood pressure and increase in pulse rate are indications for termination of emetine therapy. An electrocardiogram should be taken before initiation of therapy and on alternate days thereafter. Flattening and inversion of the T wave are changes to be expected, but widening of the ventricular complexes calls for discontinuing this drug.

Preliminary results, yet to be confirmed, suggest that glaucarubin may be as effective as emetine and has the advantages of less toxicity and oral route of administration.

Bismuth glycolylarsanilate, carbarsone, thiocarbarsone, Balarsen, diiodohydroxyquin, chiniofon, oxytetracycline, chlortetracycline, erythromycin, and Fumagillin all have been found to produce permanent cure in a high percentage of nondysenteric cases of amebiasis.[8, 15, 29, 31, 45] The preparations and dosages usually given at the Tulane Tropical Medicine Clinic are as follows: bismuth glycolylarsanilate, 250 mg. for children six years of age and under, 500 mg. for older children, three times a day for one week; or diiodohydroxyquin, 210 mg. daily per 15 pounds of body weight up to 650 mg. three times a day for 21 days. Bismuth glycolylarsanilate is the more effective but also more toxic.

In all cases of intestinal amebiasis chloroquine should be given as prophylaxis against amebic hepatitis or liver abscess. While amebic liver abscess is uncommon in children, in consideration of its gravity and the low cost and low toxicity of the drug, prophylaxis is indicated. The dose for adults and children over 10 years of age is 500 mg. twice a day for two days, then 250 mg. twice a day for 12 to 19 days. Children six to 10 years old get a 250-mg. dose for the first two days, then 125 mg., and those under six get 125 mg. twice a day for two days, then once a day for 12 to 19 days.

Surgical Aspects of Amebiasis. While surgical complications in general are rare in children, nevertheless certain features should be considered.[8, 15] As previously mentioned, right lower quadrant pain and tenderness may be the principal symptoms of amebiasis in children as in adults. The picture presented may be indistinguishable from that of chronic or occasionally even acute *appendicitis.* The surgeon should request preoperative stool examinations for all patients suspected of

having chronic appendicitis. Some will have amebiasis and may be cured medically. Appendectomy in the patient with cecal amebiasis carries an elevated risk.

Perforation of the bowel, a principal cause of death in intestinal amebiasis, is likely to occur only in the fulminating, acute dysenteric type of the disease. It is favored by clumsy proctoscopy with insufflation of air under pressure or by various enema treatments. It is prevented by early treatment for amebic dysentery. This may involve the administration of emetine if the amebicidal antibiotics fail. Once perforation has occurred, surgical intervention may be necessary, but if generalized peritonitis has been produced by free perforation, surgery is of little help. Localized peritoneal infection may be treated by drainage following preliminary administration of amebicides, including emetine.

Massive hemorrhage due to amebiasis is rare. We have never seen this complication in a child. In several adults with intestinal amebiasis who suffered massive hemorrhage, some other disease—carcinoma or idiopathic ulcerative colitis—was coexistent. Thus, when amebas are demonstrated in the stool of a patient with massive hemorrhage, the investigation should continue, since amebiasis is likely to be coincidental.

Ameboma, a firm, granulomatous tumefaction of the bowel wall due to chronic, severe intestinal amebiasis, is an infrequent complication of this disease. It tends to develop at the same levels of the large intestine as the usual amebic lesion, and may occasionally be seen in the rectosigmoid area. The principal importance of ameboma is its gross resemblance to carcinoma by inspection, palpation and roentgenologic examination. Dysentery is usually, though not always, present with ameboma. Partial resection of the bowel performed under the impression that the lesion is a neoplasm is hazardous. Preoperative stool examinations should be done routinely on all patients with a colonic mass. Those with amebiasis should receive emetine therapy, which will ordinarily cause the ameboma to decrease in size within five days. If this occurs, emetine should be followed by effective amebicidal treatment. Eventually surgery may be necessary if residual fibrous tissue produces partial or complete obstruction.

Severe chronic amebiasis with or without ameboma may result in scar formation with *stenosis.* This may develop in the rectum, but is more common in the cecal region, and may require resection of the stenotic segment or short-circuiting procedures. Surgery must be preceded by appropriate amebicidal therapy.

As previously indicated, *extraintestinal amebiasis* is rare in children, although amebic liver abscess has been reported. The latter may usually be recognized by bulging and fixation of the right leaf of the diaphragm and enlargement and tenderness of the liver. Diagnosis is established

by aspiration of the typical reddish-brown pus. Sometimes there is pleural effusion, or the abscess may rupture into the pleural space, producing empyema, or into the lung, producing pulmonary abscess and hepatobronchial fistula, with expectoration of the characteristic pus. Amebic liver abscess is treated by the administration of chloroquine in prophylactic doses, but the course is continued for three weeks or until cure is obtained. Aspiration of pus using aseptic technique may be necessary and will usually hasten cure.

Amebiasis cutis results from repeated or prolonged contact of the skin with a liquid menstruum containing many highly pathogenic amebic trophozoites. This may occur on the chest or abdominal wall at the mouth of a sinus tract leading from a ruptured abscess, or on the perineum when there is severe dysentery, with invasion and destruction of the skin and subcutaneous tissues. Treatment is directed principally at the primary amebic disease—either abscess or colitis. Administration of emetine and local application of solutions of bacitracin and chloroquine may be helpful.

Prevention

Control of amebiasis and its eventual prevention depend on acceptance of this infection as a public health problem. This concept is based on knowledge that the disease is clinically important; it is no respecter of age or race, and it is widely disseminated among urban and rural populations in temperate as well as warm climates. Attack on this problem requires active cooperation between the practicing physician and public health workers, (1) to provide safe water and sanitary sewage disposal; (2) to apprehend and treat infected persons, not only those who are seriously ill with the disease, but likewise food handlers and others responsible for transmitting cysts of *E. histolytica;* (3) to undertake chemotherapeutic control in highly endemic areas; and (4) to educate the public so that they can understand how amebiasis is contracted and how it can be prevented by improving personal and group hygiene.[15]

BALANTIDIASIS

Epidemiology

Balantidium coli, the only ciliate protozoon producing human infection, is a common parasite of monkeys and hogs. It resides in the wall of the large intestine, where it multiplies in the trophozoite stage. In diarrheic and dysenteric stools it is evacuated as a vigorously active organism; in semiformed or formed stools it is encysted. When cysts are

introduced into the mouth as a contamination, they provide opportunity for the infection to develop in individuals of the same host species from which the cysts originated. Apparently strains of *B. coli* of the hog are not easily adapted to the human host; hence human infection develops only occasionally in spite of abundant exposure to hog excreta. However, once the organism becomes established in man, it appears to be readily passed from person to person by the ingestion of cysts.[16]

Balantidiasis is occasionally found in children and adults in the United States, especially in mental hospitals, but is prevalent only in tropical climates.

Pathogenesis

After excystation in the small intestine the active trophozoites of *B. coli* are carried into the large intestine, where successful ones burrow into the wall, producing cup-shaped ulcers in the mucosa, at times extending into the deeper layers. Almost without exception these ulcers with their relatively large openings soon become bacterially contaminated; hence the early balantidial lesion resembles the chronic stage of the amebic lesion of the colon, with marginal infiltration of neutrophilic leukocytes and fibroblasts.[16]

Symptomatology, Diagnosis and Treatment

Symptoms produced by balantidial infection of the human colon vary from inappreciable to a severe dysentery indistinguishable clinically from that of amebiasis. The appearance of the bowel on proctoscopy also resembles that seen in amebiasis.

Diagnosis is by demonstration of the causative organism in the stool. This is readily made, since this large ciliate is both easy to see and to recognize, but free-living ciliates which readily contaminate unformed fecal specimens must be excluded. Spontaneous loss of the infection may occur, or treatment may be necessary.

Treatment is probably best accomplished by use of the arsenical amebicides or oxytetracycline in the same dosage as for amebiasis.[20]

Prevention

Since the risk of contracting infection from hogs or monkeys is relatively slight, methods of control in human balantidiasis should be directed to better personal and group hygiene, particularly in mental institutions, where the infection is at times endemic.

WHIPWORM INFECTION

Epidemiology

Trichocephalus trichiurus (also referred to as *Trichuris trichiura*) is exclusively a human parasite; it is prevalent and the worm burden is high in warm moist climates. This infection is common in children in endemic areas. In the South it is widely distributed, and in some children infection with this worm constitutes a serious clinical problem. In other parts of the United States it is only occasionally encountered.[16]

Each adult female worm produces daily about 9000 eggs, which are evacuated in the stool in an unembryonated state and develop to the infective stage in about two weeks if they are deposited on warm, moist, shaded ground. On being ingested as a contamination of finger tips, food, water, or as a result of geophagia, the eggs hatch in the duodenum. The escaping larvas pass down slowly to the cecal area, become attached by threading their anterior ends into the mucosa, and in about three months become mature. If there are relatively few worms in an infection, they are confined to the cecal area; but if the infection consists of hundreds of worms, they are more likely to be distributed through the entire length of the large intestine.[16]

Pathogenesis

At each site where a whipworm is attached there is pinpoint focal injection, frequently inflammation and petechial hemorrhage, together with excess mucus secretion. The delicate wiry worms become matted together in a matrix of viscous feces, mucus and extravasated blood. In heavy infections essentially the entire wall of the colon may become inflamed and hypersensitive, with occasional prolapse of the rectum.

Symptomatology

Light *Trichocephalus* infections are apt to be asymptomatic and do not usually involve the sigmoidorectal region. On the other hand, massive infection is of importance to the pediatric proctologist in areas where trichocephaliasis is highly endemic, since it produces chronic debilitating diarrhea and marked damage to the lower bowel.

The syndrome of massive whipworm infection is characteristic. It usually occurs in children between the ages of one and four years. The child fails to gain normal weight and may exhibit retarded development. The principal complaint is chronic diarrhea or dysentery, which will continue for months; if untreated, the condition eventuates in rectal prolapse.[50] Laboratory findings include microcytic hypochromic anemia and slight or moderate eosinophilia. The stool contains Charcot-Leyden crystals, abundant mucus and numerous *Trichocephalus* eggs.

In the Charity Hospital at New Orleans whipworm is more commonly a cause of dysentery in children than is amebiasis. On the other hand, amebiasis is much commoner among children with heavy trichocephaliasis than among those without the worm infection.[27]

Diagnosis

Diagnosis is based on the clinical picture and the demonstration of eggs in the stool. In order for the dysenteric syndrome to be present and for the worms to attack the rectosigmoidal area the infection must be heavy. Some type of egg count is therefore necessary to evaluate the helminthic infection as a cause of the symptoms. The simplest and most practical technique for the clinician is the direct smear method. In general, the infection is unlikely to produce important symptoms or to involve the sigmoidorectal area if there are fewer than 10 eggs, and it will usually do so if there are more than 50 eggs per milligram of feces (approximately half the amount in an ordinary 22-mm. square microscopic fecal film). However, in new infections there may be enough immature worms to produce symptoms even though the egg count is low. This situation is diagnosable only by demonstration of the young transparent worms by proctoscopy.[27]

The condition of the mucosa in whipworm dysentery visualized by sigmoidoscopy is quite different from that seen in amebic dysentery. There are diffuse "beefy" hyperemia and edema as well as tiny hemorrhagic spots possibly left by detached worms. The friable bowel mucosa is easily damaged and may bleed even when brushed with a cotton swab. Ulceration may occur, in which case the ulcers are not undermined and are mostly located along the apices of folds. The glistening white bodies of the mature worms may be seen attached to the mucosa. They are firmly adherent, and one removed by traction with forceps will break in two or take a small piece of epithelium with it. The immature worms are transparent and smaller and are easily overlooked when covered by the abundant cloudy mucus. When examined microscopically, the aspirate obtained at proctoscopy contains erythrocytes, leukocytes with an increased number of eosinophils, Charcot-Leyden crystals and *Trichocephalus* eggs.

Treatment

Since it is rarely possible to eliminate the infection completely and since light infections are usually asymptomatic, only moderate and heavy infections are routinely treated. Thus the objective of therapy is to convert the heavy symptomatic infection into a subclinical one.

Oral therapy is generally unsatisfactory, although many different drugs have been tried. The best, which is unfortunately unavailable

in this country, is the simplest, namely, *leche de higuerón*, the unmodified fresh sap of a Central and South American tree related to the edible fig.[21] The dose is 2 ounces (60 cc.) taken on an empty stomach. One should be careful to rule out allergy to this crude preparation before it is administered, since it produces severe dermatitis and stomatitis in sensitive persons.

Ferric and ammonium citrate in a dose of 6 to 12 gm. daily, though not highly efficient, will sometimes remove many of the worms and has the advantages of cheapness and relatively low toxicity. It is dispensed in 25 per cent solution in a sweet wine such as Malaga or port. The dose is adjusted to an amount just below that which produces diarrhea. Treatment is continued for several weeks.[6]

Other preparations for oral administration, such as tetrachlorethylene, emetine bismuth iodide, hexylresorcinol, gentian violet, piperazine and the broad-spectrum antibiotics, are of little value.

Heavy whipworm infection is best treated by enema. Enemas of *leche de higuerón* have been successful, but in the United States *hexylresorcinol enema* is preferable.[27] The technique is as follows: A cleansing enema is first given. Then the lower abdomen, genitalia, perineum, upper thighs and buttocks are coated with petroleum jelly to protect the skin from the burning effects of the hexylresorcinol. Warm 0.2 per cent hexylresorcinol suspension is instilled slowly until 500 cc. have been given or the patient cannot tolerate more. It is retained 30 minutes (retention is effected by holding the buttocks together), then allowed to be discharged. A small, tepid tap-water enema stimulates return of the suspension if this does not occur spontaneously.

One such hexylresorcinol enema is usually sufficient, but if mucus is overabundant, the treatment may need to be repeated. This may be done after three days and following sigmoidoscopy to ascertain the presence of residual worms in the lower bowel. The appearance of the bowel reverts to normal with remarkable rapidity (48 hours) after the worms have been removed.

Prevention

This consists basically in the sanitary disposal of human excreta, and in care for the child's personal hygiene.

OXYURIASIS (ENTEROBIASIS, PINWORM INFECTION)

Epidemiology

The human pinworm, *Enterobius vermicularis*, is cosmopolitan in its distribution and is a common parasite of children, particularly in cool

climates where wintertime bathing and change of clothing are infrequent. The infection is much more common in large families and children's asylums than in the same age group of the population at large, but is otherwise unrelated to economic status. It is likewise more prevalent among children who sleep together in the same bed or even in the same bedroom than in homes where each child has his own bedroom.[9, 16]

Typically, eggs of the pinworm are not passed in the stool. The gravid female migrates from the cecal area down the bowel and out through the anus, depositing about 9000 eggs in a meandering track on and around the anus and on the perineal skin. Occasionally in females there may be an excursion into the genital tract, and via this route into the peritoneal cavity. At the time the eggs are deposited outside the anus they are nearly embryonated and within six hours are mature. Infection or reinfection occurs from one or more of the following four methods of exposure, listed in the order of their frequency and clinical importance: (1) direct anus-to-mouth transfer of eggs on finger tips; (2) contact of fingers with clothing or other contaminated objects (table tops, chairs, toilet seats, and so on), from which eggs get into the mouth; (3) eggs temporarily floating in the air of a contaminated bedroom or school room, drawn into the mouth, especially of "mouth-breathers," then swallowed; and (4) hatching of eggs in mucus attached to the anus of an infected person, allowing the freed larvas to migrate retrograde up the large bowel to the cecal area, where they become attached and develop to maturity. Eggs which are swallowed hatch in the duodenum. The escaping larvas pass down the small intestine and on reaching the cecum or appendix become superficially attached to the mucosa, where they develop to mature males and females in four to six weeks.[16]

Pathogenesis

Although in about 2 to 5 per cent of infected persons *E. vermicularis* causes inflammation of the appendiceal wall, either by sucking blood or by opening a way for pathogenic bacteria, the common damage produced in oxyuriasis is scarification and subsequent pyogenic infection of the perineum due to unconscious scratching of the pruritic area where the worms are migrating and ovipositing.

Symptomatology

By far the commonest complaint in pinworm infection is that of anal or perineal pruritus. On close questioning, intelligent patients impart

the information that the itching more often results from a crawling or pricking sensation due to the movement of the worms in or about the anal canal than from irritation from the adherent eggs. The urge to scratch may be almost irresistible, and in some patients the perineal skin may be seriously damaged by the fingernails.

The development of neurosis in children as a result of pinworm infection is less likely than the reverse. Neurotic children are more prone to suck their fingers, bite their fingernails, masturbate and be disturbed by the crawling worms than are normal children. These symptoms of neurosis favor the building up of heavy infections.

In heavily infected girls worms wandering into the vagina may cause vaginitis. Graver consequences of the worms' migration into the genital tract, such as myometrial abscess, salpingitis and peritonitis, are rare, but should be kept in mind when a heavily infected female child presents signs of intra-abdominal or intrapelvic inflammatory disease.[48] In such cases, in addition to the usual findings of peritonitis or pelvic inflammatory symptoms, there may be an appreciable eosinophilic leukocytosis.

Diagnosis

The diagnosis of pinworm infection is based on (1) detection of worms at or emerging from the anal opening or (2) eggs collected from the perianal skin. In order to evaluate the severity of infection several specimens should be taken on successive days.[28] Information obtained from such a series also helps to establish the relation, if any, between infection and symptoms.

To obtain the perianal impression sample a 3-inch strip of cellulose adhesive tape ¾-inch wide is held sticky side out over the end of a wooden tongue depressor. The buttocks of the child are separated and the tape is applied firmly to the anus, being pressed first to one side, then the other. The tape is then smoothly spread sticky side down on a 1 by 3 inch microscope slide. After the preparation has been made the child's name and the date are written on a small slip of paper inserted under one end of the tape.

Enterobius eggs, when present in large numbers, can be seen through the transparent tape with the low power of the microscope. If the eggs are scanty or hidden in epithelial cells and fecal debris so that they are not found immediately, the tape should be marked to indicate the area to be searched, then lifted, and a drop of toluene added to clear the preparation. After adding the toluene the tape can be spread again by a firm stroke with the edge of a stiff blotter.

Tests are best made before the child arises in the morning.

Treatment

Three groups of drugs have proved effective in the treatment of pinworm infection, viz., gentian violet medicinal, piperazine and the tetracycline antibiotics.[3, 4, 28] The tetracyclines are not recommended because of cost, side effects and risk of developing strains of pathogenic bacteria resistant to these antibiotics.

Gentian violet medicinal, the standard remedy, is given in enteric-coated tablets designed to disintegrate in four hours. A dose of 10 mg. per year of apparent (not chronologic) age is given each day in two or three divided doses for seven days. Usually the drug is better tolerated if given about an hour before meals, but the timing varies from patient to patient. Side effects of treatment are nausea, vomiting and diarrhea. Nausea and vomiting may be expected if the tablet's coating is cracked. This may be checked before administration by dropping one or two tablets in water and looking for a wisp of violet color coming from them. Failures of treatment occur when the coating fails to dissolve in the intestine. This may be checked by examining the stools, which will be colored homogeneously violet if the tablets are disintegrating properly.

Piperazine hexahydrate (Antepar) given in a dose of 400 to 500 mg. three times a day for one or two weeks is also highly effective and is essentially without side effects. Occasionally it fails without apparent reason except possibly abnormal resistance of the worms. This drug is available in both syrup and tablet form.

Ordinarily only symptomatic patients are treated. Often it is discovered that the most heavily infected member or members of the family are not the symptomatic ones. In such a situation they, too, should be treated. On the other hand, it is useless to treat an entire family with the expectation of preventing reinfection within the group unless it is unusually isolated.

Prevention

Once pinworm infection has got into a household or institution, eradication is difficult or practically impossible. Simultaneous treatment of all infected persons in the group combined with strict disciplines of personal hygiene is required, but this may not suffice if another source of infection is available. Small children must be provided with closed sleeping garments so that their fingers will not come in direct contact with the itching perineal skin; all undergarments and bed linens must be frequently washed in hot water to kill adhering eggs; children's fingernails must be cut short and their hands thoroughly scrubbed each time after they visit the toilet and before meals; bed linens and night clothes should not be shaken in closed rooms, and dusting of tables,

dresser tops and other furniture in bedrooms should be done with oil-dampened cloths.[9, 16] Most families may feel that a relatively mild infection does not warrant such an expenditure of effort with little hope of permanent success, but in families of the better class, in which oxyuriasis is regarded as a social stigma, combined chemotherapy and meticulous hygiene may at times be rewarded by eradication of the infection.

STRONGYLOIDIASIS

Epidemiology

Strongyloides stercoralis is endemic in most warm humid climates of the world. Although this infection has been occasionally reported as indigenous in the northern United States and Canada, it is commonly encountered only in the coastal areas of the Gulf of Mexico and in mental institutions in the United States. Dogs harbor this nematode for only a few months, after which they lose the infection, and no other acceptable host is known to serve as a reservoir. Thus human strongyloidiasis almost invariably results from previous human infection.

Parasitic female *S. stercoralis* (parasitic males have not been found in man) live in the intestinal mucosa, most often at the duodenal level, where they oviposit. The eggs embryonate and hatch in the tissues, with the escape of active larvas into the intestinal canal. In transit down the intestine the larvas feed, grow and characteristically transform into the second feeding larval stage before they are evacuated in the stool. If the feces are deposited on warm, moist, shaded soil, the larvas rapidly transform into the infective (filariform) stage capable of entering the skin of persons who come in contact with the infested soil. Soon the larvas reach cutaneous venules and are passively carried to the lungs, where they break out of the pulmonary capillaries into the alveoli, then migrate up to the epiglottis, crawl over into the posterior pharynx and are swallowed. On reaching the duodenum the female worms enter the mucosa and in about 23 to 28 days develop into ovipositing adults. This type of life cycle is the *direct mode* of development. In tropical areas the larvas on favorable soil may produce one or more free-living generations before the larval progeny transform into the stage infective for man (*indirect mode* of development).[16]

Of much greater clinical importance is the likelihood of reinfection (*autoinfection*) by larvas which reach the infective stage at the lower levels of the intestinal canal or on the anus, without a period of development on the soil. These larvas penetrate the intestinal mucosa or perineal skin and by internal or cutaneous venous routes reach the lungs and proceed with their characteristic migration to the intestine.[17]

Pathogenesis

Tissue damage in strongyloidiasis is incidental as the infective-stage larvas enter the skin; it is much more severe as they escape from the pulmonary capillaries into the air sacs, and to an even greater degree in the intestinal mucosa, where considerable trauma occurs as a result of the movements of the parasitic females, oviposition, hatching of the larvas from the eggs and their escape from the mucosa. This may cause considerable honeycombing of the tissues and denudation of areas of mucosa occasionally as large as 1 cm. in diameter. Moreover, typically there is both local and systemic allergic reaction, with high eosinophilia.[16]

Symptomatology

Although strongyloidiasis frequently causes diarrhea or symptoms of dyspepsia and epigastric pain suggestive of peptic ulcer or chronic cholecystitis,[25] disease of the sigmoidorectal area or anal canal is not often produced. Multiple petechial hemorrhages of the mucous membrane have been observed during proctoscopy of one patient in whose protoscopic aspirate prefilariform larvas of the parasite were demonstrated. These lesions may have represented the sites of penetration of other *Strongyloides* larvas which had invaded the mucosa. A red papular rash of the buttocks, possibly of the same etiology, has been seen in children with this infection, but the relation of the dermatitis to the parasitic infection has not been proved. It has been recognized that cutaneous larva migrans (creeping eruption) may be caused by *Strongyloides*. In such cases the intensely pruritic serpiginous lesion may begin in the anal region or buttocks and extend rapidly to other parts of the body as the larvas move through the skin.[40]

Diagnosis

This consists in recovery of the larval stages of *Strongyloides* from the stool or by duodenal aspiration.[16, 25]

Treatment

Thus far no satisfactory cure has been devised for the larva migrans, although one may suspect that adrenocorticosteroids and antihistaminics may ameliorate the symptoms. Logically, treatment is directed at the intestinal infection. In our experience the best results have been obtained with gentian violet medicinal given orally on an accelerated schedule, although this treatment frequently fails. The drug in 1½-hour coated tablets is given over a four day period, with an initial dose for

children of 10 mg. per year of apparent (not chronologic) age, divided into two or three doses. Each day the dose is increased 5 mg. per year of age, until on the fourth day it amounts to 25 mg. per year of apparent age a day. The drug is discontinued whenever vomiting occurs. Administration of gentian violet medicinal (1 per cent aqueous solution) by duodenal intubation may result in cure. Intravenous administration of gentian violet is complicated, and the results do not justify its use.

Prevention

In an endemic region this requires care not to step barefooted on contaminated soil or otherwise expose the skin to ground infested with *Strongyloides* larvas. For the infected person constipation must be avoided and the anal canal and perineal skin must be cleansed of fecal contamination after each bowel movement to prevent autoreinfection.

MANSON'S SCHISTOSOMIASIS

Epidemiology

Schistosoma mansoni is prevalent in Africa and Brazil, is indigenous in the Guianas and Venezuela and in several of the West Indies, including Puerto Rico. Although it is not endemic in the United States, about 10 per cent of Puerto Ricans living in New York City, Chicago, Florida and other parts of the country harbor this parasite. Man is the only common definitive host of this parasite.

The adult worms live in mesenteric-portal blood, where the females discharge their characteristic eggs. Some of these escape through the intestinal wall into the canal and are evacuated in the stool. When they reach fresh water, the eggs hatch and the emerging ciliated larvas (miracidia) enter appropriate species of snails, in which there is a period of development and multiplication. After about one month fork-tailed larvas (cercarias) escape from the snails into the water and become attached to the skin of persons wading, bathing or washing clothes in the infested water. These larvas enter the skin, reach cutaneous blood vessels and pass through the blood stream via the lungs until they reach portal blood. Here they grow and then migrate for the most part into the smaller branches of the inferior mesenteric vein, where, about seven weeks after skin exposure, they mature and the female worms begin to lay eggs.[16]

Pathogenesis

During the incubation period, and particularly after the young worms have reached portal blood, there is increasing local and systemic reac-

tion to the toxic metabolites of the worms, most marked by painful hepatitis. When oviposition begins, eggs filtering out of mesenteric venules through the intestinal mucosa produce notable trauma, with miliary hemorrhage. Later many eggs become trapped in the intestinal wall and in increasing numbers in the periportal tissues, provoking pseudotubercles around the eggs, fibrous strictures and papillomas of the colon, and portal cirrhosis. The pathologic process is the more serious because it builds up gradually for 30 or more years during the life of the worms.[16]

Symptomatology

To the proctologist schistosomiasis mansoni is of particular interest when the stage of deposition and extrusion of eggs is reached about seven weeks after skin exposure. Because of the chronic inflammation and ulceration around the eggs in the wall of the bowel, an intermittent dysentery is produced which is characterized by cramps and the frequent passage of bloody mucus. Attacks of dysentery are commonly provoked by exercise. As the disease progresses, hepatomegaly and eventually cirrhosis occur.

In these patients the rectum appears to have a thickened mucosa with submucosal hemorrhages. There may be numerous bleeding ulcers and miliary abscesses, an abundance of mucus containing eosinophils, macrophages and usually the characteristic lateral-spined eggs of the parasite. The peripheral blood typically presents an eosinophilia, and there may be anemia due to chronic blood loss.

The stage of tissue repair is characterized by the production of papillomas of the colon and rectum, the wall of which is scarred, thickened and sometimes constricted. Although dysentery usually subsides in this stage, the damaged bowel is subject to ulcerative degeneration.[44]

Diagnosis

In patients with relatively light infection the disease may produce only mild symptoms. In the absence of characteristic symptoms diagnosis may be difficult. It is based on stool examination, rectal biopsy and immunologic tests, in addition to the clinical picture. Stool examination should include studies not only of direct smears, but also of sedimented specimens or concentration by other methods.[16] Rectal biopsy[19] consists in snipping a small portion of mucosa from one of Houston's valves. The tissue is compressed and examined under the microscope for the lateral-spined *Schistosoma* eggs. Immunologic tests include intradermal, complement-fixation, agglutination and precipitation techniques, all of which are adjuvants in establishing the diagnosis, but should not be relied on alone.

Treatment

Potassium and antimony tartrate (*tartar emetic*) is the most effective drug in the treatment of schistosomiasis. It is administered intravenously as a freshly prepared 0.5 per cent solution in 5 per cent glucose or isotonic saline solution. An adult receives initially 8 ml. (0.04 gm. of tartar emetic), and on alternate days thereafter the dose is increased by 4 ml. (0.02 gm.) until a daily level of 28 ml. (0.14 gm.) is reached. A total of 18 doses, amounting to 444 ml. (2.2 gm. of tartar emetic), constitutes a course of treatment.[36] For children the dose should be reduced according to body weight. In these patients considerable difficulty is usually experienced with thrombosis of the veins and extravasation of the irritating solution of this drug.

Fuadin (stibophen), which is given intramuscularly, is preferred for children, even though its cure rate is lower than that of tartar emetic. On the first day 0.5 ml. of a 6 per cent solution is administered into the gluteal muscles. The dose is increased by steps to 3.5 ml. and continued at this level on alternate days until a total of 15 to 25 ml. has been given.[36]

Prevention

This may be accomplished by one or more of the following methods: (1) sanitary disposal of the excreta of infected persons; (2) avoidance of exposure by keeping out of water in endemic areas; (3) using chemicals in water in endemic areas to kill the snail intermediate hosts;[51] and (4) treating all infected persons until they no longer discharge the fluke's eggs in their stools. None of these methods has been notably successful.

Other Forms

In extensive areas of the world children suffer from infection with two other species of blood flukes. In China, Japan and the Philippines *Schistosoma japonicum* produces a disease syndrome similar to that of *S. mansoni*, but with more rapid development, because of the fact that this species of blood fluke lays approximately tenfold as many eggs daily as does *S. mansoni*. In highly endemic areas the disease frequently results fatally before the child reaches 16 years of age. The third species is *S. haematobium*, which is indigenous to almost the entire continent of Africa, the Near and Middle East, southern Portugal and India near Bombay. The eggs are laid in the rectal venules and in the vesical plexus and are discharged respectively in the stool and urine, with dysentery and hematuria.[16]

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1430 Tulane Avenue
New Orleans, Louisiana

THE ADMINISTRATION OF ENEMAS TO INFANTS AND CHILDREN

GLADYS S. BENZ, R.N.

An enema is a liquid introduced into the rectum or colon. Enemas may be classified as (1) those expelled immediately or within a matter of minutes after administration, and (2) those retained for long periods of time, varying from an hour or more to the time necessary for complete absorption.

Liquids administered rectally include plain water, suspensions, mixtures or true solutions. Because of common usage the term "solution" will be used in the following pages to designate all liquids given rectally even though the liquid is not chemically a true solution.

Although tap water and soap are commonly used as ingredients for cleansing enemas, there is considerable evidence that this practice is not entirely safe.[4, 5, 7, 8, 9, 12] Rapid absorption of water may produce hypotonicity of the extracellular fluid and water intoxication. Although the risk of this is greatest in the presence of megacolon, it has been known to occur when the bowel is normal.[1] Isotonic saline solution is easy to make and is substantially safer as a routine vehicle for enemas in children. Even diagnostic barium enemas are preferably made up in isotonic saline solution, especially if megacolon is suspected. In the presence of megacolon combined with renal or cardiac impairment, agar solution or 7 per cent gelatin solution has been recommended as a cleansing agent.[4, 8]

Enemas are administered for cleansing, diagnostic or therapeutic purposes. The nurse should understand the purpose of the physician's request and do everything possible to help achieve the desired results with a minimum of trauma to the child.

From the Department of Nursing, Cincinnati Children's Hospital.

CLEANSING ENEMAS

The purpose of a cleansing enema is, as its name implies, to clear the bowel of fecal material. When used as an adjunct toward asepsis (as for proctoscopic examination or double contrast x-ray studies using barium and air), such cleansing needs to be especially thorough. It need not be quite so thorough when used as a preparation for anesthetic or preanesthetic agents given by rectum, for removal of barium after x-ray examination, or for the elimination of gas and feces prior to pyelography. The degree of effectiveness of a cleansing enema may determine the success or failure of the procedure which follows it.

DIAGNOSTIC ENEMAS

Diagnostic enemas are used primarily in roentgenologic studies and usually consist of liquid suspensions of radiopaque materials such as barium sulfate. In certain circumstances air is injected for added visual contrast. Diagnostic enemas represent an intrinsic part of radiologic techniques and need not be reviewed here. Under certain conditions, such as the evaluation of abdominal pain due to constipation or fecal impaction, an ordinary cleansing enema may have diagnostic value.

THERAPEUTIC ENEMAS

Therapeutic enemas are used for such a wide variety of purposes that even a condensed summary of the indications for them would needlessly prolong this review. Even a cleansing enema may have therapeutic value. Suffice it to say that the composition and quantity of the solution used depend entirely on the specific therapeutic indication for which the enema is given. These, for example, may include enemas of cool or iced fluids to reduce high fever, oil retention enemas as an adjunct to the removal of fecal impactions, certain drugs when other routes of administration are not feasible at the time, or for barium suspensions (both diagnostic and therapeutic) for the reduction of intussusception.[2, 10]

PREPARATION OF THE CHILD

There is always the possibility that an enema may be something of an unpleasant or trying experience. There may be a little discomfort, some fear and, with the adolescent, even some embarrassment. The approach should be kind, patient and without hurry, with plenty of time for explanation in terms that the child can understand. It may be well to let the child handle the equipment and feel the solution for warmth. Liken the insertion of the rectal tube to the insertion of a rectal ther-

mometer. The child may fear pain if this is his first enema or if he has been hurt or uncomfortable during previous ones.

Resistance may be caused by pain, fear, temperature of the solution, excessive amount of solution or too rapid administration. Abdominal relaxation may be encouraged by preparatory explanation, by having the child lie on his side and breathe deeply, and by giving the solution slowly without force or excessive pressure. A second person may be needed to assist the small child in maintaining a satisfactory position. The child should, if possible, sense that he is actually helping, thus avoiding a feeling of antagonism or rebellion toward the person and the treatment. The child who realizes that he can give up or retain stool at will and thus control adult concern for him, may feel attacked or punished. Something is being taken from him without his consent. Such a child needs even more careful preparation, explanation and assurance that he is helping. The cooperation of the child is of prime importance.

An older child may be allowed to help control the speed of flow of the solution. He must be assured that the flow will be slowed or interrupted if he becomes unduly uncomfortable. He will be more likely to cooperate if he is assured in advance that his feelings will be respected and if he knows that he can depend upon the nurse for truthful explanations and answers to his questions. A cheerful and helpful attitude on the part of the nurse will favorably influence the child's reactions. When a cleansing enema is given prior to barium contrast study, it often is desirable to explain to the child that he will have another rectal tube inserted in the x-ray examining room. If he is old enough to understand, he should be told in advance something about the x-ray examination, since cooperation is difficult in the dark and unfamiliar setting of that procedure. The nurse who administers the cleansing enema can contribute substantially to the success or failure of the x-ray examination.

PRECAUTIONS IN THE ADMINISTRATION OF ENEMAS

The nurse must keep abreast of current theory and practice so as to be able to perform her function in enema administration intelligently and effectively. Her understanding of how she influences the success of the treatment and how she can assist the child will help her realize why an enema should be administered with great care. The nurse should understand the reason for the procedure, the desired effect to be achieved, and the potential hazards involved.

Since water pressure increases with the height of the water, it has been recommended that the base of the reservoir of solution should not

be more than 4 inches above the rectum.[11] Fluid will flow into the rectum by gravity when there is a difference in height between the solution in the container and the end of the outflow tube, unless the contents of the colon preclude the introduction of more fluid. If pressure is too great, there may be pain and a desire for immediate evacuation. The rate of flow of fluid is influenced by the inside diameter of the tube and the viscosity of the fluid used.

The rectal catheter should be well lubricated to reduce friction as it is inserted. An exception to lubrication may be indicated in certain

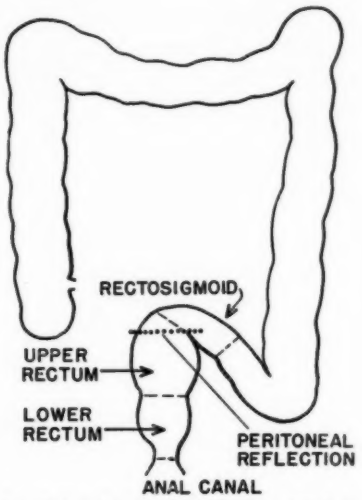

Fig. 41. The rectal catheter should be gently inserted just far enough to pass it through the anal sphincter. Perforation of the bowel can occur in the area of the peritoneal reflection if force is used in attempting to pass the catheter beyond that point. (M. Cook, after Nelson: Textbook of Pediatrics, 6th ed.)

instances, such as the reduction of intussusception with barium, when it is important that the tube be kept in place through the entire procedure. There is evidence that a rupture of the colon can be mechanically produced by the tip of the enema tube.[6] This should serve as a precautionary warning to the nurse to insert the tube gently, just far enough to pass it through the anal sphincter unless a greater distance is specified by the physician (see Fig. 41).

Fluids used in enemas should approximate normal body temperature of 98° to 99° F. unless otherwise specified by the physician. The usual deviation is in cool or iced solutions for the reduction of body temperature. The amount of solution given should be measured. If approxi-

mately the same amount is not returned, it may be necessary to siphon off the fluid. Any failure to expel the solution, any untoward effects of the treatment or failure to achieve its purpose should be brought to the attention of the physician.

Flow of solution should be interrupted if the child shows signs of distress. The buttocks may be pressed together or elevated on a pillow to help him retain the solution long enough to be effective. Slight pressure over the anus with a folded diaper may help the child retain the solution during intermittent periods of discomfort.

The child old enough and well enough to sit up may be supported in this position while the solution is expelled. Even an older child should not be left completely alone or unsupervised for any length of time during the expulsion of an enema. He can be given privacy and the nurse can still be within call, or she can return frequently to observe his condition. The small infant will be more comfortable if he expels the enema into a diaper or pad rather than being held over a container. The child should be removed from the bed pan as soon as he feels he is finished. The young child who cannot tell whether expulsion is complete should have a rubber protector and pad left under him after the diaper has been replaced. He should be praised for his accomplishment and be left clean, dry and comfortable. His hands should be washed and the unit aired if necessary. If a stool specimen is needed for laboratory examination, a sample should be set aside.

CONTRAINDICATIONS TO THE ADMINISTRATION OF ENEMAS

Contraindications to the administration of an enema may be relative or absolute, and much depends upon the judgment of the physician who orders the procedure. The only absolute contraindications are the presence of existing, suspected or potential perforation of the bowel. The last of these three is especially important in the postoperative period following resection and anastomosis in the large bowel or rectum.

JUDGING THE EFFECTIVENESS OF THE TREATMENT

The nurse should be able to evaluate the effectiveness of the treatment. Asking herself a few questions may be of help in such evaluation. Was the procedure safe? Was the child comfortable? Was embarrassment prevented? Was exhaustion prevented? Were the purposes of the treatment achieved? Was skillful workmanship evident throughout? Was the recording accurate and pertinent? Did the child feel a sense of achievement? When properly administered, an enema need never be a traumatic or frightening experience.

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Elland and Bethesda Avenues
Cincinnati 29, Ohio

PSYCHOSOMATIC ASPECTS OF PEDIATRIC PROCTOLOGIC LESIONS

CHARLES BRADLEY, M.D.

In common with disorders of other parts of the body, a number of diseases of the lower intestinal tract are recognized as being largely influenced by psychologic factors. The term "factor" is an important one. Few, if any, proctologic disorders are known to be the result solely of psychologic stress. However, emotional factors may loom so large in the background of some that the success of their treatment may hinge on their psychologic management.

Among disorders of the colon and rectum which are particularly influenced by emotional stress in children are infantile colic, some forms of diarrhea, encopresis (involuntary soiling with feces), constipation and ulcerative colitis. Mucous colitis, which would ordinarily be included in this list in adults, is practically unknown in childhood.

EMOTIONAL STRESS AND PHYSICAL SYMPTOMS

Before pointing out some of the salient psychologic features of these several disorders, a few comments may be made as to how emotional stress may find expression in physical symptoms. More extensive and penetrating discussions of the subject are scattered throughout the literature of psychiatry and psychosomatic medicine. Unfortunately, no comprehensive review of pediatric psychosomatic problems has yet appeared.

When physical symptoms result from psychologic stress, their origin is usually to be sought in emotional conflict. By this is meant that at least two incompatible sets of feelings are striving for supremacy and

From the Department of Pediatrics, University of Oregon Medical School, Portland

expression in the same person at the same time. The physical symptoms are a by-product of this struggle. This may be illustrated by what is said to occur in many men who suffer from peptic ulcer. These men, even in advanced adult life, have a deep basic need for mother love, protection and even nursing. Such feelings are of course incompatible with the conventional concepts of masculinity which most men apply to themselves. Men unconsciously try to deny these dependency needs and attempt to compensate for them by strenuous efforts to achieve material success and social prestige whereby they can prove to themselves and to the world that they are mature, masculine fellows. Their gastric mucosa is more responsive to their basic needs than to their conscious wishes and reacts by hypersecretion as though it were awaiting the maternal nourishment which never comes. As a result, hyperacidity and its various sequelae contribute to what we know as the ulcer syndrome. Meanwhile the victim continues to strive for accomplishments which can never really satisfy his fundamental needs. If one believes with the pragmatist that all symptoms serve useful purposes, it is comforting to assume that at least the ulcer symptoms tend to slow the patients down.

Psychosomatic symptoms referable to the lower intestinal tract follow the same sort of principles. In childhood they usually involve conflicting feelings toward persons who are of greatest importance to the child. These are usually, of course, his parents and siblings, and perhaps most frequently his mother. His conflicts regarding her usually stem from mixed feelings of love, need or desire for her on the one hand, and fear, hate or hostility directed against her on the other. Conflicts may of course originate elsewhere, but when they do, they are apt to be less intense and certainly occur less frequently.

In psychiatric literature the functions of body structures are often described as symbolizing certain activities which meet the patient's emotional needs. Interpretations of colitis are a case in point. Here the coordinated actions of several portions of the gut are involved in a symptom commonly described as diarrhea. This may be regarded as symbolizing rejection, expelling or "getting rid of and doing away with" something. Such interpretations are clinically helpful. No individual symptom, however, should be accepted as prima facie diagnostic evidence of some particular underlying emotional conflict.

Now let us briefly consider the several disorders which are the main subject of this discussion.

INFANTILE COLIC

Colic is a rather broad term used to describe what appear to be recurrent attacks of severe abdominal pain in certain young infants. Though there is no proof that this originates in the colon or rectum,

it at times so resembles the symptoms of obstructive lesions in these organs that the proctologist may be called in consultation. There is likewise no clear-cut proof that the disorder is of strictly psychologic origin. However, many pediatricians are impressed by the frequency with which it occurs in infants whose mothers are tense and overanxious.

Colic is essentially a disorder of the first six months of life. The infant may appear lusty and in good health, but periodically appears to suffer acute abdominal pain which may persist from 30 minutes to as long as four hours. Some observers feel that his cry at such times has a characteristic quality which distinguishes this particular type of distress. Attacks may be precipitated by feeding, and when they occur, the infant may draw his knees up over his abdomen. While the colic may be relieved by the passage of flatus, true distention is not a distinguishing feature. At times the baby's distress may be so intense that it is accompanied by sweating and pallor. Though this may suggest transitory intussusception, vomiting and other evidences of obstruction are not conspicuous. A tight anal sphincter, an inguinal hernia, an intercurrent urinary infection or a food allergy may in some children be related to the occurrence of colic, but none is recognized as its usual etiology. Fortunately it tends to disappear with the passage of time as the infant grows older.

That anxiety on the part of the mother is transmitted to the colicky infant by the way in which she handles him is certainly a possibility. On the other hand, the infant's distress is sufficient to produce anxiety in most mothers. One can only speculate as whether anxiety is a cause, an effect, or the link that completes a vicious circle in this disturbing symptom complex. In any event, measures which will bring relief to both mother and baby are important therapeutic considerations.

In the writer's experience, mothers of children later coming for study to a child psychiatry clinic report an early history of colic in their children with unusual frequency. Such histories, however, show no definite correlation with the occurrence of any specific psychologic symptoms later on.

In the light of our present knowledge, incomplete as it is, *treatment* of colicky infants should include due consideration for the mother's emotional state. The capable clinician will facilitate treatment by accepting the mother's feelings as part of the problem and by giving her support, encouragement and reassurance, realizing that she cannot change her feelings at will.

CONSTIPATION

Though constipation may not be a problem of the same magnitude in children that it is in adults, it is encountered frequently enough in

pediatric practice. By common consent among pediatricians, emotional factors are usually of more importance than structural defects in its genesis. Constipation in infants may be related to the make-up of their diet, but is less likely to be the case in older children. Moreover, correlation between a history of constipation in infancy and its occurrence later is not conspicuous. Family patterns of frequency of bowel evacuation vary considerably. What is considered a medical symptom by one family is passed off as inconsequential by another. Constipation is more apt to be a problem in children when parents focus a great deal of attention upon it.

Most infants, toward the latter part of their second year, go through a phase in which they are temporarily intrigued with their excretory functions. Playing with urine and feces and interest in their production are quite natural at this time, however much it may dismay parents, who can rarely be complacement about such activities. Most mothers capitalize on this spontaneous interest by instituting "toilet training" at this time, and ordinarily their efforts meet with success. However, at this very same age the alert, healthy infant is also much concerned with establishing himself as an individual. One of his favorite devices in asserting himself involves refusal to cooperate with whatever is requested of him. This may include obstinate resistance to moving his bowels when and where his parents want him to. The result may be constipation, and its emergence as a persistent problem is apt to hinge to a considerable extent on the amount of emotional turmoil stirred up by the baby's resistance to training.

Emotional overtones may be added to the whole business of toilet training by a variety of cultural factors to which the physician must be alert. These range from traditional superstitions about autointoxication to current emphasis on the prompt and regular removal of waste from the human body amid the splendid surroundings of the most gorgeously equipped chamber in the home—the modern bathroom. Many mothers sorely need the understanding and sympathetic assistance of the physician in helping them maintain a casual air of equanimity regarding their baby's toilet habits, and in proportion as this evolves, the threat of constipation diminishes. Homely rituals, such as increased fluid intake, extra bulk or fruit in the diet, an adequate bathroom time for the child, all help the mother carry out a positive approach to the problem, for which no panacea is at hand. If little children seem fearful of flush toilets (a very real threat for some), a comfortable "potty chair" in another room may help.

Elimination of constipation may of course not be as simple as some of the foregoing comments suggest. They have been made to emphasize the sort of psychologic factors which may contribute to this problem

and which must be dealt with by the physician above and beyond purely "physical" considerations. So-called spastic constipation seems to be less of a problem in children than it is in adults. Perhaps this is because most youngsters are less under the pressure of modern civilization, which stimulates many adults to maintain themselves constantly poised for "fight or flight," with consequent disruption of gastrointestinal tract function.

The following case report illustrates some of the psychodynamic considerations involved in one seriously constipated child.

CASE HISTORY. Pete was an 8 year old boy referred for psychiatric treatment because of generally poor social adjustment with other children, severe temper tantrums and infrequent bowel movements associated with apparent emotional distress and physical pain. The latter symptom had been present since infancy, when he had failed to respond to "toilet training" about 18 months of age. His bowels moved about once a week. He seemed acutely uncomfortable for several hours preceding the performance of this function. A large amount of feces resulted, and the toilet was frequently stopped and required a plumber's attention after his defecation. He was an only child, whose mother had lost a previous baby in infancy with amyotonia, the child apparently choking and suffocating to death in her arms. She had been very apprehensive of Pete's health since his birth, and throughout infancy he had recurrent attacks of bronchitis and croup which terrified the mother. She was a capable person otherwise, active in community affairs, running her home with warmth, enthusiasm and efficiency, but tended to react to her boy with many symptoms of oversolicitude. At the time they came to the clinic the mother was still cleaning the child after his bowel movements in the bathroom. She was also giving him his baths and washing him. The father was a somewhat passive man who had been seriously constipated for many years, but was able to control this with reasonable attention to his diet.

In the course of several weeks of therapy for the child and several months of guidance for the mother, the boy was enabled to achieve more independence and the mother confidence in letting him be much freer from her domination. Aside from her ceasing her activity of personally cleansing him after bowel movements, no attention was paid to this particular symptom, which gradually decreased until 9 months after starting contact with the clinic the boy had an evacuation every day or two with no accompanying distress or complaints on his part.

In this family the boy had become the object of extreme oversolicitude by the mother as a result of her tragic experience with her older child and the fact that she had been sensitized to his health because of his intercurrent upper respiratory illnesses in infancy. His training in bowel function was presumably carried out inconsistently and with a great deal of attention from the mother, who continued to supervise it until the time he came to clinic attention. The major therapeutic efforts in this case were directed towards helping the mother understand the nature of her problem, relieving her anxieties regarding the boy, and helping her relinquish her domination over him. As he gained in poise and self-confidencee and she was able to let him assume responsibility for himself, his constipation diminished until it was no longer a problem.

DIARRHEA

Diarrhea apparently occurs less frequently in children than in adults as a symptom of emotional stress alone. In particular, it is not often encountered as a primary or secondary symptom in children who come to the attention of a child psychiatry clinic. Some of the literature on the emotional significance of diarrhea is misleading, for it confuses the symptom with encopresis or the initial stages of ulcerative colitis. These are more accurately dealt with as entities in their own right. Diarrhea alone may be by-passed in a brief discussion such as the present one.

ENCOPRESIS

Encopresis is a term coined by Weissenburg in 1926 to describe fecal soiling. This is primarily a disorder of childhood and is analogous to enuresis. It is less frequently encountered than enuresis and further differs from the latter in that it commonly is a diurnal rather than a nocturnal affair. Encopresis is more common in boys than in girls.

The usual story is that of a child who soils or stains his clothing at intervals with semiformed or loose stool. Encopresis has come to be recognized as essentially a "behavior symptom" and usually appears at times of stress in youngsters who have other difficulties in social adaptation. Because of the associated problems of odor and cleanliness, it is particularly objectionable to the adults who are in close contact with the child. Such soiling is easily interpreted by them as a deliberate and willful performance carried out to attract attention or as an act of hostility or aggression. Careful study of children with reasonable sensibilities invariably reveals them to be more distressed by their soiling than are their parents or fellows, and indicates that they are entitled to sympathetic clinical consideration.

The literature on encopresis has been thoroughly reviewed by Lehman,[2] who stresses the psychologic nature of the problem and suggests ways of meeting it. It is essentially a symptom of regression. This means that certain children who are more or less chronically emotionally dissatisfied with their present surroundings attempt to console themselves by falling back on behavior patterns which were part of their normal life in infancy. It is as though those "good old days" when mother met all one's needs and there were no personal responsibilities were happier than the present time, and any reminder of the earlier period is pleasurable. The child is, of course, not aware of this basis for his behavior. His parents naturally find it hard to recognize, and even harder to accept, fecal soiling as a means of consolation for anyone. Thumb-sucking is an analogous symptom which is easier to understand.

Regressive symptoms like encopresis which involve somatic activity or a physiologic "habit" are notoriously resistant to direct *treatment.*

Extensive medical, laboratory or roentgenologic procedures serve to establish more firmly, rather than to relieve, the problem. Encopresis should be dealt with as casually as the personality of the parents and the emotional atmosphere of the home permit. Therapeutic efforts should be focused on whatever may give the child increasing satisfaction in his day-to-day experiences, whether this be a more secure position in the affections of his family, remedial work in certain key school subjects, or greater opportunities for success in his physical activities and social contacts. Psychiatric consultation or child guidance treatment has a definite place in the rehabilitation of many children with encopresis.

Case Histories

The following two patients may illustrate some of the mechanisms involved in the cause and treatment of this disorder.

CASE I. Robert was a 6 year old boy who was admitted to a children's psychiatric hospital because of failure to respond to treatment in a community child guidance clinic for soiling himself with feces 5 or 6 times daily since infancy. His father was 45 years of age when the boy was born and had previously lived in the home of an older sister who had both dominated him and met all his needs. His wife was somewhat younger and came from a large, happy family, but had been very much concerned with her own bowel functions since she had been a young girl. The mother had placed a great deal of emphasis on her attempts at toilet training prior to 2 years of age, and the boy's first response had been to appear stubbornly constipated, later shifting to encopresis. The home atmosphere was tense, the parents being sexually and socially incompatible. The father's older sister frequently interfered with the parents' attempts to deal with the youngster. Physical findings in the hospital were entirely negative.

In the nursery school group the boy's general social reactions were those of a younger child, with considerable hoarding and inability to share his belongings with others. He rapidly gained social poise, and the mother had frequent psychotherapeutic interviews during the several months the boy remained in the hospital nursery school. She was able to work through much of her feelings about her unhappy marriage, but was most obsessive about her preoccupation with her own bowel functions and her concern about her boy. In spite of improvement for both mother and child during the hospital period, the home situation continued very much the same, and a follow-up note on Robert when he was 12 years of age indicated that occasional soiling during the day in school was still a problem. In this case the boy appeared to be the victim of an unhappy marriage complicated by interference by paternal relatives and his mother's extreme preoccupation with bowel function.

CASE II. Martha was a 13 year old girl who had been soiling her clothing with feces during the daytime intermittently since the age of about 5 years. Her parents' other complaints about her were her diurnal and nocturnal enuresis, occasional stealing both at home and in the community, and general incompatibility with her younger sisters, aged 11 and 8. Her mother is said to have died from colitis when the patient was 5 years of age and shortly before the onset of Martha's behavior difficulties. The father had remarried when Martha was 7 years of age, and both parents had found her morose, unhappy and frequently negativistic to the family routines. Her school progress had been fairly satisfactory.

Because of her parents' difficulty with her, she was admitted for study and treatment to a children's psychiatric hospital, where from the day of admission she made an excellent adjustment and there was no evidence of any of the symptoms which

had been such a problem at home. She was somewhat older than the other children in the hospital group and rapidly and capably assumed responsibility for assisting with nursery school children and helping some of the children in the lower grades with their academic work. Psychotherapy and guidance for both Martha and her parents was carried out coincidentally over an 8 week period. A follow-up report several years later indicated no recurrence of symptoms. This girl had apparently reacted to deprivation of her own mother and jealousy of her younger sisters both by reverting to infantile eliminative habits and stealing as an act of aggression. The parents' attempt to deal with her in a firm and somewhat impersonal way had not supplied her with basic desires and dependency needs. When in the hospital these could be met, and later with guidance her parents could meet them, there was no longer need for her symptoms.

ULCERATIVE COLITIS

Ulcerative colitis occurs from earliest infancy to advanced old age, and most pediatricians of experience will have encountered it occasionally in their practice. Many clinicians feel that psychologic factors have considerable importance in this disease, and there is an extensive literature on this aspect of the subject. This has recently been reviewed by Engel,[1] who notes that numerous workers consider that the diarrheal aspects of this disorder symbolically express the patient's desire to expel, reject or discard something unacceptable to him. For many patients the initial symptom is bleeding, and the appearance of the classic manifestations of colitis is often preceded by a variety of types of disordered lower intestinal tract function, including constipation. This leaves some question as to whether one should assume that the whole disease process invariably expresses a deep-seated desire to be rid of psychologic material which is repulsive to the patient. Regardless of what conclusions one draws, there is little doubt that most patients with the disease are involved in serious emotional conflicts which precede and often precipitate their clinical symptoms.

Prugh[3] has reported a careful study of the personality of children who suffer from ulcerative colitis, and many of his findings are consistent with what others have reported for adults. He notes the presence of unresolved, largely unconscious, feelings of anger on the part of the child, which is directed towards persons by whom the child wishes to be loved. One would anticipate that these persons would most frequently be the parents. The onset of symptoms is usually shortly preceded by some disturbing precipitating event. There are often circumstances which may have something to do with the child's choice of the lower intestinal tract as the site of his symptoms, such circumstances being illustrated by disturbance of bowel function in one or both parents. The child may achieve some of his deep-seated desires, such as those for extra love, attention or solicitude from the parents, by becoming ill. Children who suffer from ulcerative colitis appear to have difficulty clearly expressing their emotions, and their outward appear-

ance, even during some of the more distressing manifestations of their illness, may remain incongruously calm, passive, subdued and even more resigned than one would expect. With recovery from the colitis they may be noted to express their feelings more freely.

The results of psychologic *treatment* of some patients are most rewarding. Results are of course best for those whose disease has not progressed to the stages at which irreversible anatomic changes have taken place. Most important elements in this treatment appear to be assistance for the child and his parents in making decisions, planning a definite program of diet, medication and activities which imposes as few restrictions as are consistent with his physical well-being, and enabling the child to gradually express his repressed feelings with the warm, accepting support of the therapist. This is an assignment for an experienced psychotherapist who can work with the entire family as well as with the pediatrician.

The true cause of ulcerative colitis is not understood at present. It is quite unlikely that psychologic factors alone are responsible. However, in certain children psychologic factors may be sufficiently important and sufficiently accessible for relief as to make them the logical focus of the treatment program. The following case report may illustrate how this was handled in one family.

CASE HISTORY. Betty was an 11 year old girl who had been having frequent stools intermittently for 2 years. There had been bouts of bleeding and increased periodic bouts of diarrhea requiring hospitalization on 3 occasions. Shortly before she came to our attention definite x-ray evidence of ulcerative colitis had been noted. After a month's hospitalization with confirmation of the diagnosis, bed rest, dietary restrictions and some antibiotic treatment, she was discharged to her home at a time when she was having 2 to 4 moderately formed stools daily. Psychiatric guidance was sought for follow-up supervision.

Betty was the eldest of 3 children, who had shown little reaction to the birth of a brother when she was 4 years of age, but had always shown marked antagonism to a sister born about a year prior to the onset of the first symptoms of colitis. Betty had never been affectionate with her parents, in marked contrast to the 2 younger children, but had always sought to be the center of attention. She was a hard worker and successful student in the school situation, but did not make or hold friends for any length of time because of her egocentricity and dominant ways.

The father was a young, noncommunicative man who worked steadily in a position which his wife thought was beneath his capabilities. He spent all his spare time with his family and in the home, often sitting reading and sometimes not speaking for hours. The mother was a vivacious, active woman who was a highly efficient housekeeper, and expressed her feelings openly. She showed her dissatisfaction with her husband's quietness and expressed her anxiety about her daughter's illness very freely in the home. She was particularly confused as to what procedure to follow with Betty. An atmosphere of veiled dissatisfaction, uncertainty and anxiety permeated the home.

Over a period of 8 months following the child's discharge from the hospital the mother was seen in weekly interviews for general guidance and reassurance. The child was checked casually in the office occasionally for the assurance of the mother and the physician that her general condition was good, but extensive clinical investigation was studiously avoided in order that anxiety not be provoked. Betty was rapidly introduced to a much more active program than she had enjoyed for 2 or 3 years.

The mother handled this with increasing finesse and assurance, and as a result of her own therapeutic interviews was able to accept the family situation, including her husband, with more enthusiasm. Betty was still doing well on a full program of activities a year after her hospitalization.

In this case the mechanisms of attention and conflict were not obvious on the surface, but apparently Betty's resentment of her younger sister, her feeling that she did not get attention from her parents, her jealousy of her younger siblings, and possibly conflict in the home atmosphere due to the father's extreme reticence, contrasted with his wife's much more vivacious character, all tended to create tension which was reflected in the girl's colitis. Betty's excellent course under calm, matter-of-fact supervision tended to confirm these impressions regarding the importance of psychologic factors in her illness.

CONCLUSION

Though undoubtedly a great deal more could be said about important psychosomatic relations in pediatric proctology, the preceding brief discussion may best be considered an introduction to the subject. An attempt has been made to describe some of the more common psychologic mechanisms involved, as well as to list a few of the more prominent syndromes in which they become manifest. It is to be hoped that more definitive discussions of all psychosomatic problems in childhood will soon be forthcoming.

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3181 S.W. Sam Jackson Park Road
Portland 1, Oregon

RECTAL BLEEDING IN INFANTS AND CHILDREN

C. EVERETT KOOP, M.D.

Rectal bleeding is a common sign in pediatrics and one which is often the cause of real anxiety to the patient's family and to his physician. Most rectal bleeding comes from local, transient lesions and is short-lived, but rectal bleeding may also herald serious hidden pathology. Because it is the physician's obligation to differentiate between the serious and the inconsequential, this clinic will be devoted to a consideration of the various etiologic lesions responsible for rectal bleeding as well as a suggested method of study directed toward proving the etiology of the bleeding in a given case.

HISTORY

There are several fundamental questions which must be answered before any evaluation of rectal bleeding can be undertaken:

1. How much blood has been passed?
2. What color is the blood?
3. Is the blood mixed with the stool or is it purely a surface phenomenon?
4. How long has the patient been bleeding?
5. Is the blood passed before, during or after the bowel movement?
6. Are there any associated signs or symptoms which might help to elucidate the diagnosis?

Although a knowledge of the quantity of the blood that has been passed as well as the duration of the symptoms is helpful in arriving at a diagnosis, one must not feel secure in the hope that he is dealing

From the Surgical Clinic, Children's Hospital of Philadelphia, and the Harrison Department of Surgical Research, University of Pennsylvania School of Medicine, Philadelphia.

with an insignificant lesion if the symptoms are chronic and the amount of blood passed is small. Children may have an exsanguinating hemorrhage as the first sign of rectal bleeding, or they may bleed for months or even years in small quantities before catastrophic blood loss is encountered. Any discussion of the amount of blood passed by rectum in the pediatric age group must take into consideration the circulating blood volume of the patient. What may be an inconsequential loss of blood in an adult may be a hemorrhage in a small infant. For example, considering an infant's blood volume to be roughly 100 ml. per kilogram, the loss of 30 ml. of blood in a 6½ pound baby is equivalent to a hemorrhage of over 600 ml. in a 150 pound adult.

The color of the blood passed by rectum, whether it is bright red or dark, gives additional information. One can assess to some degree closeness of the bleeding lesion to the anus as well as the rapidity of the passage of blood to the outside from the lesion. The action of acid on blood to produce the brown coloration of acid hematin indicates that the blood has been in contact with hydrochloric acid in the stomach, a duplication or a Meckel's diverticulum. Bright red blood usually indicates a rectal or at least a colonic lesion, although it is possible to have a large volume of blood come down from the small intestine so rapidly that it appears at the anus bright red in color.

Blood found only on the surface of the stool indicates a lesion low in the rectum where mixture of fecal material and blood has not been produced by colonic peristalsis. The time relationship of the passage of blood in reference to the passage of stool is of some help in diagnosis when taken together with other facts elicited in the history.

Perhaps more important than any other sign except that of profuse bleeding is that of any evidence of associated phenomena such as abdominal pain, vomiting, nausea, cramps or the picture of shock. Silent bleeding from a lesion such as Meckel's diverticulum or a duplication of the bowel may necessitate in itself immediate operation. Abdominal pain or vomiting, or the onset of shock accompanying bleeding even in the absence of profuse hemorrhage, may be an indication for surgical intervention in some infants, and at least alerts the physician to the suspicion that the sign of rectal bleeding is the signal of a serious pathologic state.

CAUSES OF BLEEDING FROM THE RECTUM

It is difficult to outline the causes of melena without having it appear like a recitation from a medical student's list of differential diagnoses. However, there seems to be no alternative to the physician's attempt to consider from mouth to anus the causes of bleeding in order that none be overlooked and the proper therapy instituted in a given case.

We have used an oversimplified chart (Table 4) for teaching medical students in an attempt to group the causes of rectal bleeding so as to show those which commonly are associated with the passage of dark blood in the stool as contrasted with those showing bright blood. As has been stated, the grouping of dark blood-producing lesions usually indicates a high intestinal lesion, whereas those producing bright red bleeding are commonly indicative of a lower intestinal lesion; yet some small bowel lesions can produce bright bleeding if blood loss is large and peristalsis particularly active. Some lesions producing melena are

TABLE 4. *Outline of Causes of Bleeding from the Rectum in Childhood*

DARK BLOOD	1. Swallowed blood		
	2. Swallowed foreign body		
	3. Esophageal varices		
	4. Peptic ulcer (pain?)		
	5. Duplication of bowel		
	6. Meckel's diverticulum		
	7. Mesenteric thrombosis	PAIN	
	8. Volvulus		
	9. Intussusception		
	10. Systemic disease		
	11. Polyposis		
	12. Neoplasm		BRIGHT BLOOD
	13. Colitis		
	14. Polyp		
	15. Chronic recurrent sigmoid intussusception		
PAIN	16. Inserted foreign body		
	17. Fistula in ano		
	18. Fissure in ano		
	19. Hemorrhoids		
	20. Rectal prolapse		

also accompanied by pain, and these have been indicated diagramatically in the table. No effort has been made to correlate other associated signs and symptoms.

The various lesions to be considered here will be discussed, not in the order of frequency or importance, but rather in anatomical order from mouth to anus. Little will be said concerning therapy of the various lesions, inasmuch as this symposium is devoted to the management of such problems. The question of when to do a laporatomy, because it presents such a difficult decision, will be discussed in some detail.

Swallowed Blood

Swallowed blood may appear at the anus after delivery, epistaxis and operations on the nose and throat. Usually the child will also vomit blood, and this knowledge aids one in arriving at a diagnosis.

Attention has been called to the appearance of swallowed maternal blood as a bloody stool in the neonatal period. Frequently there is a

history of placenta praevia or some other obstetric complication with or without fetal distress that raises a suspicion as to the etiology. A simple qualitative test described by Apt and Downey[1] is useful in differentiating the grossly bloody stool caused by gastrointestinal bleeding from that contaminated by maternal blood.

Swallowed Foreign Body

Most parents and some physicians think of this cause first when a child bleeds from the rectum. Actually it is a rare cause for blood in the stool, and many foreign bodies of considerable size and unusual sharpness are swallowed without causing any melena. Bleeding produced by gastrointestinal trauma is usually small in amount and dark in color. It is associated intimately with the stool and is not accompanied by other signs unless there has also been perforation of the bowel. When bleeding from the bowel can be associated by history and x-ray diagnosis with an ingested foreign body, removal of this object by endoscopy or laparotomy is usually indicated.

Esophageal Varices

Bleeding from esophageal varices is almost always associated with portal hypertension, and concomitant signs of hepatomegaly, splenomegaly, collateral circulation and ascites as well as a preceding history of hepatitis or jaundice are of considerable help in making a diagnosis. Most patients who bleed from esophageal varices also vomit blood, and the diagnosis becomes relatively simple. The underlying medical problem must be adequately managed and then consideration given to the surgical procedure most likely to relieve the hypertension in the portal system.

Duplication of the Bowel

Duplications of the gastrointestinal tract can be found at any level from esophagus to rectum, but the most common site for a bleeding duplication is the small intestine. The cause of bleeding is usually a marginal ulcer occurring at the site where normal small bowel epithelium is in contact with aberrant gastrointestinal epithelium, as is frequently found in Meckel's diverticulum. In the over-all occurrence of melena this is an unusual cause for bleeding, but on an active children's service it is seen frequently enough as the cause of painless, brisk, bright red bleeding from the bowel to be mentioned here. Such a duplication should be removed surgically. Emergency laparotomy is of course indicated when the volume of bleeding is alarming. Under

such circumstances exact diagnosis is usually impossible until after the abdomen has been opened.

Peptic Ulcer

Bleeding from the bowel due to peptic ulcer is almost as uncommon in childhood as it is common in adult life. However, real hemorrhage from gastric or duodenal ulcers following massive burns, head injuries and intracranial operative procedures occurs often enough so that one must be aware of the diagnostic possibility. Most bleeding from peptic ulcers in childhood is small in amount, and some may be detected only as occult blood in the stools. Such patients respond well to medical treatment, but operation is occasionally lifesaving in the patient with an acute ulceration followed by brisk hemorrhage.

Meckel's Diverticulum

The cause of bleeding from Meckel's diverticulum or omphalomesenteric duct is caused by ulceration of the mucosa where the alkaline secreting intestinal mucosa meets the acid-producing diverticulum. Meckel's diverticulum is found frequently in comparison with the more rare duplication and should be thought of any time when there is painless bleeding from the bowel.

Such bleeding is usually small in amount, but can be massive. Most Meckel's bleeding is bright red, but dark blood is also possible. Both inflammation and fistula formation with an adjacent loop of bowel may occur in association with the bleeding, so that inflammatory signs of parietal peritoneal irritation or other symptoms are also possible.

Meckel's diverticulum may be the cause of an exsanguinating hemorrhage and death. Therefore if, by elimination, Meckel's diverticulum is suspected as the cause for bleeding from the bowel, exploratory laparotomy should be undertaken. This is frequently an emergency procedure.

Intussusception, Volvulus, Mesenteric Thrombosis

In these problems intermittent pain or a shocklike syndrome is more commonly the cause for medical concern than is bleeding. Rarely it is the bleeding which raises one's suspicions to otherwise vague symptomatology. Fear of irreversible compromise of the blood supply to the bowel demands immediate surgical therapy except in those instances in which barium enema reduction of intussusception seems feasible and safe.

Systemic Disease

The physician dealing with a child one of whose complaints is that of bleeding from the bowel, must bear in mind constantly that the cause of such bleeding may be other than surgical. Blood dyscrasias, allergy, nutritional disturbances due to lack of vitamins, acute and chronic infections, parasitic infections and absence of one or more of the blood-clotting elements are among the possible etiologic factors.

The rectal bleeding in such instances is usually not profuse, may be in or on the stool, and either bright red or dark. Other stigmata of the underlying disease can aid in diagnosis.

Polyps and Polyposis

Polyps of any variety occurring in the large bowel may bleed during childhood. Such polyps may be single or multiple, and rarely one sees true multiple polyposis, usually on a familial basis. In the latter instance the danger of malignant change is far greater than that from bleeding alone and is sufficient indication for colectomy. Multiple polyps must not be confused with multiple polyposis.

Most bleeding caused by polyps is bright red, on the surface of the stools, intermittent in character, and occasionally passed without accompanying feces.

Colitis

A great many children pass some blood during attacks of diarrhea which produce a concomitant colitis which may be nonspecific in origin. Such bleeding is also seen from specific causes such as amebic or ulcerative colitis.

Milk allergy is not an infrequent cause of bright red streaking of the stool in infancy and is usually accompanied by colicky pain. Examination of the rectal mucosa through a proctoscope shows it to be congested, and bleeding occurs on slight trauma.

Medical management of all types of colitis is indicated with the exception of surgical measures necessary in the advanced or fulminating cases of ulcerative colitis.

Chronic Recurrent Sigmoid Intussusception

Occasionally in a chronically constipated child who repeatedly strains at stool a recurrent type of sigmoid intussusception takes place without compromise to the blood supply.[2] Such a lesion is a real source of bleeding. This bleeding is bright red, on the surface of the stool, and small in amount. Proctoscopic examination reveals the bleeding tip

of the intussusception, and the nature of the lesion cannot be appreciated without the use of an adult-size proctoscope.

Daily digital dilatation of the rectum for several weeks with the use of an emulsifying agent to soften the stool accompanied by bowel training usually takes care of the situation.

Neoplasms

Both benign and malignant neoplasms, including hemangiomas, may occur in the colon and higher and produce bleeding from the bowel, although such lesions are extremely rare in the age group under discussion. However, no medical consideration of the child with melena is complete without considering this possibility.

Inserted Foreign Body

This is a rare cause for rectal bleeding which is bright red in character and frequently painful. Diagnosis is usually confirmed by rectal examination.

Rectal Causes of Rectal Bleeding

Because of the nature of the accompanying papers in this symposium, nothing will be said in detail concerning fissure, fistulas, hemorrhoids and rectal prolapse.

STUDY AND DIAGNOSIS

One soon learns which patients require an emergency type of management and which can be studied at relative leisure. As has been said previously, hemorrhage is always a cause for immediate action, and hemorrhage in infants may be represented by what seems to be an insignificant amount of blood.

Under ideal circumstances we believe that patients should be seen jointly by a pediatrician and a surgeon and that such studies, including red blood cell count, hemoglobin concentration and cross match for transfusion, should be undertaken immediately. Bleeding and clotting time as well as prothrombin time and occasionally a study on fibrinogen levels is also indicated. Proctoscopic examination is next in order except in those instances in which it is obvious that the bleeding must be coming from some point higher than can be seen through the rectum or in which hemorrhage is so brisk that laparotomy is indicated rather than proctoscopic examination. X-ray examination is of help only on rare occasions in the emergencies. When the history reveals

the bleeding to be chronic or intermittent and the amount of blood is small, additional blood studies are sometimes undertaken and barium enema with air contrast and upper gastrointestinal x-ray series carried out. It should be remembered that x-ray studies practically never reveal Meckel's diverticulum when present and seldom show a duplication.

Careful observation of patients with intravenous fluid replacement and frequent repeated blood studies are in order until diagnosis is accurate and the decision concerning laparotomy has been made.

While awaiting diagnosis and a decision concerning therapy, blood loss should be replaced by transfusion if the amount of loss so indicates. The danger of exsanguination in most children and infants is so great that in the younger age group we believe the indications for laparotomy are more liberal than in larger children. Although tragedies are rare, shock from hyposalemia and death occur often enough so that those who see many pediatric patients with melena have a real fear of the consequences of bowel hemorrhage.

It is inevitable that the surgeon who performs many laparotomies for bleeding from the bowel will occasionally encounter patients who have no demonstrable lesion to account for the blood loss. Such findings should not necessarily influence the surgeon to do fewer laparotomies, because he performs the valuable service of ruling out a lesion like hemangioma, Meckel's diverticulum or duplication of the bowel which, untreated, could bring about the death of the patient.

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18th and Bainbridge Streets
Philadelphia 46, Pa.

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